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ARCHIVES OF OPHTHALMOLOGY.

CONCERNING PAPILLEDEMA IN TUMORS OF THE BRAIN AND ITS SURGICAL TREATMENT.[1]

By Dr. CHARLES A. ELSBERG, New York City.

(With twelve illustrations on Text-Plates XIII.–XV.)

"DROPSY of the sheath of the optic nerves," said de Wecker almost fifty years ago, in one of his celebrated lectures in the Rue du Cherche midi, "dropsy of the sheath of the optic nerves may exist either with intracranial pressure or with inflammation of the papilla." In those days no clear distinction was made between papilledema and optic neuritis, but in many respects de Wecker's statement is applicable to our knowledge of today. We have learned a great deal since de Wecker's time, and better appreciate the intimate connection between advancing intracranial disease, and changes in the fundi and that the ophthalmologist has an important rôle in the diagnosis and hence in the treatment of intracranial disease.

Our experiences at the New York Neurological Institute have shown us that great aid can be given to neurologist and surgeon by the expert in ophthalmology who has a full understanding of neurological problems and their association with changes in the fundus oculi. Dr. Holden has—based on his large experience—presented to us his views on the differences between true papillary edema, and inflammatory lesions—a distinction which is often not well appreciated. True optic

[1] Read at the December, 1923, meeting of the Section on Ophthalmology of the New York Academy of Medicine.

neuritis is an inflammatory lesion which one often sees in toxemias, in intracranial suppuration and in sinus disease. It is an entirely different condition from what occurs as a result of a rise in intracranial pressure.

Everyone is agreed that papilledema—whether it occurs early or late in the course of intracranial expanding lesions—must and soon will be considered a *late* symptom of brain tumor.

One has, only, to compare the very large size of many tumors removed at operation (see Fig. 1) with the eye findings that were observed in the patients, in order to realize how long the growths must have existed before they caused any fundus changes at all. The majority of patients come under our observation when they already have more or less swelling of the discs; often when the edema has reached a high grade with considerable diminution in visual acuity. The individual with a tumor of the brain should come to the surgeon when the changes in the eye grounds are slight and often when there are no fundus changes at all. This is already the case in tumors in the posterior cranial fossa,—especially many that originate from the sheath of the auditory nerve, in the cerebello-pontine angle. During the past few years, I have seen and have operated upon a constantly increasing number of these patients before there was any evidence of papilledema, and I am convinced that the best results can be obtained in this early stage.

I. PAPILLEDEMA IN TUMORS OF THE POSTERIOR FOSSA AND ESPECIALLY IN TUMORS IN THE CEREBELLO-PONTINE ANGLE.

The history and the physical signs of the so-called angle growths are usually so typical that if the patient came under observation early enough, the correct diagnosis should be made and the surgical interference done, before disc changes have occurred.

The proper time for surgical treatment—from the standpoint of the patient and hence from that of the neurologist and the surgeon,—is before the tumor has become so large that it directly or indirectly causes pressure upon the aqueduct of Sylvius with a resulting hydrocephalus and papilledema, and before the growth has reached a size large enough

ILLUSTRATING DR. CHARLES A. ELSBERG'S ARTICLE "CONCERNING PAPILLEDEMA IN TUMORS OF THE BRAIN AND ITS SURGICAL TREATMENT."

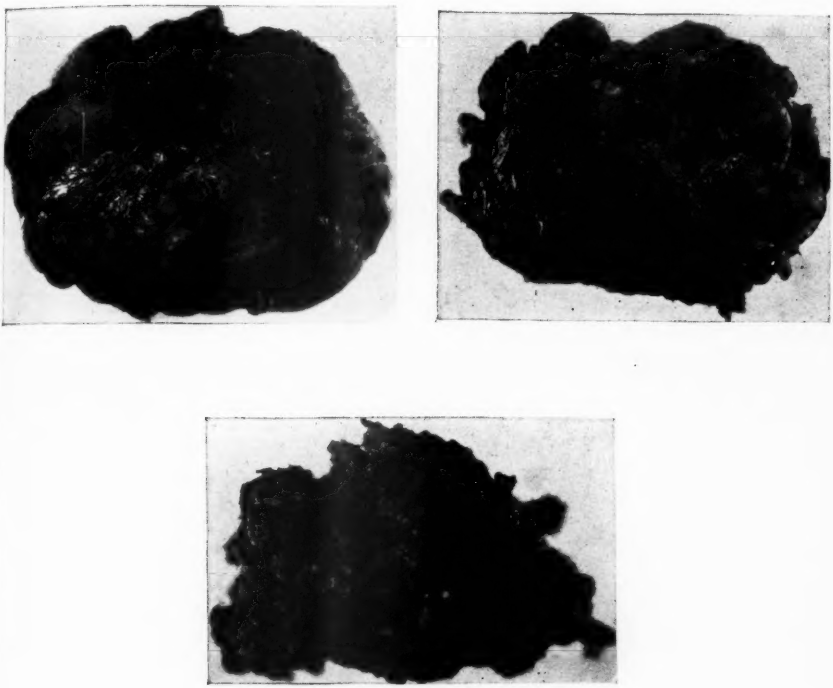

FIG. 1. Showing very large brain tumors removed from patients who had only a slight degree of papilledema.

to impinge upon and often to become adherent to the side of the pons and medulla.

Until this lesson is brought home, the number of cases of cerebello-pontine angle tumor in which complete extirpation of the growth is possible will be far below what it ought to be.

It has been our experience that in these angle growths, the appearance of the papilledema may be sudden and its progression very rapid. Thus I have seen and operated upon a patient who had been carefully watched for almost a year on account of left angle tumor symptoms. After twelve days, the fundi, hitherto normal, showed a swelling of 3 diopters in each eye, which increased to 6 diopters on the left and 5 diopters on the right, with many hemorrhages and $\frac{20}{100}$ vision in the left and $\frac{30}{100}$ in the right eye in the following four days. In another patient, the change from normal fundi to a swelling of 4 diopters occurred in one week.

These instances,—which could be multiplied—show that the onset and progress of the swelling may occur rapidly.

It is a fact for which I should like to have an explanation, that in these lateral recess tumors, the papilledema is often most advanced in the eye homolateral to the tumor. Thus, in the last 30 patients that I have operated upon, 19 had more swelling on the same side as the growth; in five, the swelling was equal in the two eyes; in six, there was no papilledema.

If the mechanical theory of the cause of papilledema is the all sufficient one, then it is difficult to understand why a tumor, which causes a ventricular distension practically equal on the two sides, as a result of compression of the aqueduct of Sylvius,—should not be associated with a papilledema with the same elevation on the two sides.

2. PAPILLEDEMA IN SUPRATENTORIAL GROWTHS.

As compared with tumors in the posterior cranial fossa, the mechanical changes in intracranial pressure due to supratentorial growths are of a more complicated character.

In expanding lesions of the cerebral hemispheres, the degree of swelling of the discs on the two sides varies greatly. The increase of intracranial pressure is due to a combination of factors,—the amount of internal hydrocephalus, the degree of hyperplasia of the affected hemisphere (Spiller) (Hirn-

schwellung of Reichard), the actual size of the tumor, and perhaps other causes. The reason why, in growths in the hemispheres, there is so much variation in the degree of papilledema on the two sides, is that the factors above mentioned may be combined in a variety of ways.

The papilledema on the same side as the tumor may be caused to a great extent by the pressure of the tumor itself, as the lateral ventricle on that side may be collapsed and the amount of hyperplasia of the hemisphere not great. On the other hand, the hyperplasia of the lobe may be the most important factor (see Fig. 2). The degree of papilledema on the side opposite to that of the tumor, may be and often is due to the distension of the lateral ventricle of that side, for a distension of the contralateral ventricle is very frequent in unilateral growths (Fig. 3). If the local pressure conditions are greater on the side of the tumor, then the papilledema on that side is the more advanced; if, on the other hand, the local pressure in the contralateral lobe is greater on account of the ventricular distension, then the fundus of that side will show the most marked changes.

A different condition obtains in some cortical and subcortical tumors of the frontal lobes. In these patients, it is not rare to find a pallor of the disc or even a primary optic atrophy on the side of the tumor, and a papilledema of a slight degree on the opposite side. The importance of these changes, from the standpoint of localization of the tumor, is very great. Such a combination of fundus changes is rare in any other intracranial lesion, altho it is occasionally seen in pituitary growths with defects in the temporal fields, where there is a large suprasellar growth and where a papilledema in one eye is superimposed upon a preceding optic pallor.

These fundus changes—unilateral pallor of the disc with contralateral papilledema—from an expanding lesion in one frontal lobe are sometimes mistaken for alterations in the eye grounds due to disease of the ethmoid sinuses, and as a result, operative interference for the intracranial lesion is too long delayed. The differentiation between a true intracranial expanding lesion, and disease of the air sinuses may be very difficult, as the latter may cause many signs of disturbed neurological function. The fundus changes may predominate

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FIG. 2. To illustrate extensive hyperplasia of one cerebral lobe associated with temporal lobe tumor.

FIG. 3. Illustrating the collapse of the homolateral and dilatation of the contralateral ventricle in a right hemisphere tumor. Contralateral hydrocephalus is very frequent in unilateral hemisphere growths.

in the clinical picture, and I have seen several patients who had lost almost all vision before the true nature of their disease was recognized.

The subject of unequal swelling of the discs in intracranial disease, has in the past occupied the attention of numerous writers, and many attempts have been made to correlate the eye findings with the location of a tumor in one or the other cerebral hemisphere. In addition to what has been stated above, I venture to say the following: Tumors of the cerebral hemispheres that lie furthest away from the median line,—and this is especially the case in cortical growths over the convexities of the brain—more often cause a papilledema which is equally advanced in the two eyes, while in tumors nearer the midline—as in many subcortical growths—the swelling of the discs is more advanced in the one or the other eye. Mesially placed growths, such as those which arise from the falx cerebri, from the pineal gland, or those rather frequent ones in the interpeduncular space most often cause changes in the eye grounds that are equally marked on both sides. Tumors in the most anterior parts of one or the other frontal lobe may reach a considerable size before any disc changes occur, and dural growths over some part of the motor area may cause unilateral convulsive seizures and slowly progressive loss of strength in the extremities of the same side long before any changes in the fundi have occurred. This was well shown in the case illustrated in Figure 4, a patient with a history of Jacksonian epilepsy of one year's duration with gradual diminution in the power of the right upper and lower extremities. She was admitted and operated upon when she was in a state of status hemiepilepticus and an endothelioma removed from the left arm area.

Finally, attention must be called to the fact that papilledema may be absent in some cases of brain tumor because the growth has compressed a large cerebral artery so that there is an extensive secondary softening. I have operated upon several patients in whom the clinical picture was that of a vascular degenerative lesion without any changes in the fundi. In one of the patients, the diagnosis of cortical tumor plus vascular degeneration was made before the operation (see Fig. 5). In another patient, an extensive softening was found

at the operation. The individual recovered from the surgical procedure and several months later,—during the last few weeks of her life,—developed a papilledema. At the post-mortem examination, a large subcortical infiltrating tumor was found which had caused secondarily a very extensive cerebral softening. It is important therefore to remember that papilledema may not occur or may occur only in the terminal stages of the disease, in these cases of brain tumor with extensive vascular softening.

We are all interested in the important question: What can surgery do for the relief of papilledema and the resulting visual disturbances? It is well to realize that if vision has once been lost from choked discs, the patient will remain blind. It has been my experience that even if the tumor is removed or a decompressive operation is performed within the first few hours after the loss of sight, no vision—not even light perception—will return.

Equally pathetic is the condition of the patient with a high grade of choked disc and marked failure of vision, in whom the tumor has been successfully removed, but in whom so much post-neuritic atrophy has occurred that the little remaining eyesight is lost within a few weeks after the papilledema has subsided and the tumor symptoms have been relieved. In not a few patients with a papilledema of a moderate grade of severity, operative interference is delayed for many weeks because of $\frac{1}{10}$ vision, and the case only considered urgent, when vision has begun to fail.

The whole professon should understand that a papilledema, —even of slight degree and without any diminution in vision— is a very serious matter which demands relief as soon as possible. If operative interference is done early—whether by removal of the tumor, a well executed decompression, drainage of a possible abscess or of infected sinuses—blindness can be prevented in the majority of instances.

A question that is of interest to you as ophthalmologists is this: How rapidly does a papilledema subside after the removal of a tumor of the brain? Without giving you any definite statistics, I can sum up my experience as follows:

In tumors of the cerebellum the papilledema usually begins to recede rapidly, so that within one to two weeks there is

ILLUSTRATING DR. CHARLES A. ELSBERG'S ARTICLE "CONCERNING PAPILLEDEMA IN TUMORS OF THE BRAIN AND ITS SURGICAL TREATMENT."

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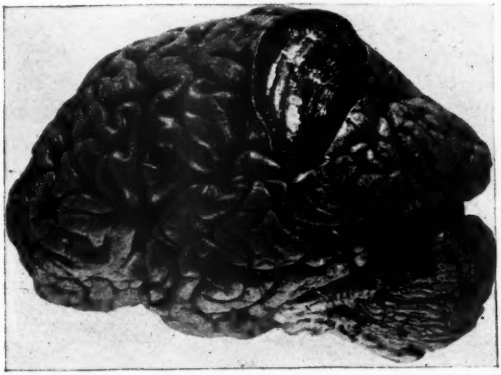

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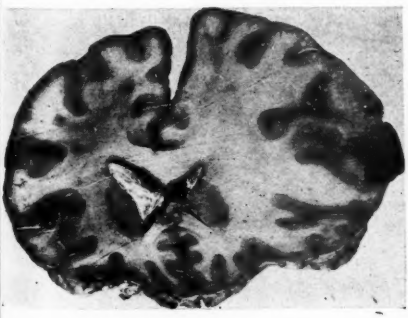
FIG. 4. A case of endothelioma over the left arm area causing Jacksonian epilepsy without changes in the fundi.

A. Photograph of a normal brain to illustrate location of the growth.
B. The growth that was removed.
C. Photograph of the patient eight days after the operation.



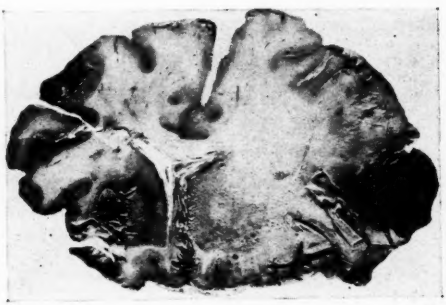


FIG. 5. Showing extensive degeneration in one lobe from a tumor in the Sylvian fissure which compressed the middle cerebral artery.

considerable diminution in the elevation of the discs (2 to 3 diopters) and a rapid absorption of preëxisting hemorrhages. Thereafter, the recession of the fundus changes is more slow, and several months pass before the discs have become flat, the exudates absorbed, and the disc margins again sharply defined. If, however, the tumor cannot be removed, the fundi will not improve unless the mechanical conditions are such that the tumor will move away from the median line and the block to the passage of cerebrospinal fluid out of the ventricular system is relieved. The evacuation of the contents of a gliomatous cyst will often accomplish this purpose very satisfactorily.

It has been claimed by some, that the decompressive effect of a suboccipital craniotomy with wide opening of the dura, is marked in only a small number of the patients. This has not been my experience. The early improvement has usually been so distinct that the absence of improvement for several weeks, has led me to suspect that the growth was fixed or that it involved also the midbrain.

After the satisfactory removal or intracapsular enucleation of a lateral recess growth, and often after the decompressive effect of the suboccipital craniotomy, with the reëstablishment of the circulation of the cerebrospinal fluid through the ventricular pathways, the papilledema will generally recede. In most instances if the swelling has been considerable—3 or more diopters—it generally requires several months before the discs are flat and the hemorrhages have been entirely absorbed. Unless there has been an undue amount of trauma to the cerebellum, the improvement in the eye grounds after the operation is very marked at the end of a week—a reduction of several diopters of the elevation of the discs and the beginning absorption of the hemorrhages. Thereafter the progress is more slow.

In supratentorial growths, on the other hand,—for the reasons that have already been explained,—the recession of the fundus changes is more gradual. In a small number of the patients, the papilledema rapidly improves so that a moderate swelling of the discs has entirely subsided within a few weeks. In most of the patients, however, the improvement is slower and proceeds at a fairly constant rate.

In several of my cases, the improvement did not begin for several weeks, and even then it was so slight for several months, that we gave some consideration to the possibility that the patient might have a second growth in some other part of the cranial cavity. This retardation in the improvement of the fundi is unusual but it occurs sufficiently often to make it worthy of mention.

What result may be hoped for, as far as the papilledema is concerned, in those cases—unhappily so frequent—in which the intracranial growth cannot be removed, and where an operation for the relief of pressure is the best that the surgeon can offer?

Several years ago, an eminent neurologist read a paper before a national society with the title "Shall we decompress for choked discs," and he expressed the belief that the reduction of intracranial pressure by a properly performed decompressive operation was rarely followed by improvement in the papilledema and in visual acuity.

With such a statement my experience does not agree. Altho the indications for decompressive operations are becoming more and more limited because the neuro-surgeon is making more frequent efforts at radical extirpation of intracranial growths, a properly placed decompressive operation will in the majority of instances, have a marked beneficial influence upon the fundus changes.

Excepting in those patients who came to operation when the papilledema was so far advanced that they were already blind or that the post-neuritic atrophy would be followed by the loss of the little vision that remained, and with the exception of some cases of irremovable midbrain tumor in which no decompressive operation is satisfactory—and these are the unfortunates in whom death is rarely long delayed—in the larger number of other patients, eyesight can be preserved by a properly timed and properly executed decompressive craniotomy.

Of course, a subtentorial decompression for a supratentorial tumor will do no good. Nor can a subtemporal decompression have much if any effect upon a choked disc secondary to an obstructive hydrocephalus from a growth in the posterior cranial fossa. In the latter type of case, decompressive operation in the subtemporal region simply permits the lateral

ventricle to become more and more dilated as it forces the brain tissue out of the cranial cavity through the opening in the skull. In such a case, a subtemporal decompression should never be done.

One might expect that if the dilatation of the ventricular cavities could be relieved by an operation by which a new pathway for the escape of the cerebrospinal fluid was made, the ensuing reduction of intracranial pressure would be followed by a subsiding of the disc swelling. With this object in view, the operation of puncture of the corpus callosum was devised, and I have seen some beneficial results from this surgical procedure. Very often, however, it has failed to accomplish the desired result in the hands of most of those who have tried it.

Are there any temporizing procedures other than a decompression that may be used, in patients with a high grade of papilledema and marked deterioration of vision, during the time that is required for the study of the case, for laboratory tests, and perhaps until air has been injected into the ventricular cavities and X-ray pictures taken? Repeated lumbar punctures with withdrawal of spinal fluid are sometimes useful, but it is well to remember that this procedure has its dangers if the tumor is located in the posterior cranial fossa, on account of the possibility of sudden prolapse of part of the cerebellar lobes into the foramen magnum with resulting pressure upon the medulla.

If there is marked ventricular distension, repeated withdrawal of fluid from the ventricles through a small trephine opening may be tried.

The use of concentrated solutions given intravenously (5 to 15 per cent. of normal salt or 25 per cent. of glucose or dextrose solution) may be very useful as a temporizing procedure. We have become accustomed to give the patients one ounce of magnesium sulphate by mouth every four hours,—if the patient can swallow, or three ounces of the salt dissolved in six ounces of water by rectum, every four hours, if it cannot be given by mouth. As is well known, these concentrated solutions often diminish intracranial pressure and therefore should, temporarily at least, relieve the pressure upon the optic nerves of the fluid retained in the sheaths of the nerves.

PAPILLEDEMA: ITS SYMPTOMS, DIAGNOSIS AND COURSE.[1]

By Dr. WARD A. HOLDEN, New York City.

THE fact which Dr. Elsberg and I wish to emphasize is that true papilledema, due to increased intracranial pressure in cases of brain tumor, leads almost invariably to complete and permanent blindness unless a decompressive operation or removal of the tumor is done early. The patient must be urged to submit to operation as soon as visual disturbances appear, for he will not consent sooner unless headache and vomiting make operation imperative. With further delay vision will be reduced permanently, and even after a successful operation is done and the papilledema relieved, the atrophy of the optic nerve will often increase weeks later, and the development of new central or peripheral defects in the field will diminish the remaining vision still further.

Lack of time since this program was arranged has prevented me from analysing my eye records of the hundreds of patients with papilledema on whom Dr. Elsberg has operated at the New York Neurological Institute. Hence I shall merely summarize the present generally accepted knowledge of papilledema in its clinical aspects.

In cases of brain tumor with prolonged intracranial pressure and dilatation of the intervaginal space of the optic nerves, the discs become edematous. With the ophthalmoscope the nasal, upper and lower margins of the discs are seen to be blurred first, and later the temporal margin. As the edema increases, the physiological cup is obliterated and the disc becomes elevated from three to five dioptries above the

[1] Read at the December, 1923, meeting of the Section on Ophthalmology of the New York Academy of Medicine.

normal level of the retina. Since the retina about the disc also becomes edematous and swollen the true elevation of the disc is measured by comparing the refraction of the surface of the disc with the refraction of the retinal vessels some disc-diameters away. A mere note of the refraction of the surface of the disc gives one no information whatsoever.

Because of the peculiar relations of the disc with the sheaths of the optic nerve in myopic eyes, papilledema in myopic eyes is a very great rarity, and the absence of papilledema in myopic eyes does not exclude increased intracranial pressure. And papilledema does not develop in an atrophic nerve, although a papilledema that has subsided without atrophy may recur with a fresh increase in intracranial pressure.

The edematous disc retains a pinkish color, and both the acuteness of vision and the limits of the visual field may remain normal for months or even years. The blind spot is enlarged, however, either because of the edema of the retina adjoining the disc, or because of the compression of the peripheral fibers of the optic nerve—a condition that was pointed out by Herman Knapp in a paper read before the American Ophthalmological Society as long ago as 1870.

After a time, periodic obscurations of vision occur and soon after, in the typical case, the field becomes contracted on the nasal side with some reduction of central vision, then a general concentric contraction ensues, encroaching upon the fixation point and later leaving only a small eccentric field of vision to the temporal side, which eventually is lost altogether. With failure of vision the disc gradually becomes pale and the swelling subsides, but the veins long remain dilated and tortuous and the disc margins blurred.

The course of the deterioration of vision is not always typical, for it may begin with sector-like defects, loss of a large portion of the upper or lower half of the field, or central scotoma. Mandelstamm in 1866 spoke of binasal hemianopsia in cases of papilledema, but Leber in 1877, in the *Graefe-Saemisch Handbuch*, considered the designation "hemianopsia" improper, since in true hemianopsia, of whatever type, central vision is normal or nearly normal. Cushing, however, has recently revived the term "binasal hemianopsia," applying it to fields of atrophy of the optic nerves following papil-

ledema, with marked contraction of the nasal half of each field.

Other forms of defect in the fields of papilledema are caused by direct pressure on the visual fibers at some point behind the orbit and are to be regarded as complications. Thus, pressure on one optic nerve just anterior to the chiasm from a frontal tumor may produce a central scotoma; pressure on the chiasm, bitemporal defects in the fields; and pressure at any point further back, homonymous defects.

Although papilledema long remains a pure, non-inflammatory, edematous process, advanced cases in which vision has become affected may show true inflammatory changes under the microscope. And some of the central scotomas are doubtless due to a late inflammation in the optic nerve.

With increase of intracranial pressure the spinal fluid also is under pressure, as is shown when lumbar puncture is done. Papilledema occurs with nephritis when there is intracranial pressure, and this papilledema diminishes when spinal fluid is withdrawn or even disappears after repeated withdrawals. Papilledema, usually of low grade, occurs very frequently with the increased intracranial pressure caused by hemorrhage after fracture of the skull.

The papilledema of pseudo-brain tumor, by which is meant acute internal hydrocephalus, is of rapid onset with early disturbances of vision, but the prognosis as regards vision is relatively good. It is caused by inflammatory processes blocking the channels of exit from the third or the fourth ventricle. The diseases bringing it about are serous meningitis, lethargic encephalitis, polioencephalitis and the like, and even, in rare instances multiple sclerosis. The spinal fluid is under high pressure and lumbar puncture may lessen the symptoms.

The so-called pseudo-neuritis of high hyperopia is congenital, stationary, of slight elevation and the blind spot is not enlarged.

Sharply to be differentiated from papilledema is papillitis. Papillitis is a true inflammation of the disc, as distinguished from the pure edema of papilledema. These two terms, now generally adopted, make it possible to discard forever the awkward "choked disc," "ascending" and "descending" neuritis, and the rest.

In papillitis the disc is swollen, but its elevation does not often exceed two dioptries and the swelling extends farther out into the retina than in papilledema. Papillitis is usually a local condition and mostly unilateral, while papilledema is but one of a complex of symptoms and is bilateral. The causes of papillitis are nasal sinusitis, inflammations of the orbit, syphilis, multiple sclerosis, meningitis (in which one eye is involved after the other), and toxic conditions such as wood-alcohol poisoning, in which both eyes are involved at the same time.

Visual disturbances often come on early in papillitis. The typical changes in the field are first, enlargement of the blind spot and, second, central scotoma which may extend until total blindness ensues. The light sense is diminished in papillitis, but not in papilledema. With papillitis, when not due to meningitis, the spinal fluid is not under pressure, as it is in papilledema.

The diagnosis of papilledema is easy when headache, vomiting and other symptoms accompany the swelling of the discs. But papilledema is, in some cases, an early and isolated symptom, perhaps discovered accidentally by the ophthalmologist in the routine examination of a patient's eyes. Then, in order to make a positive diagnosis, extensive research may be necessary. The sensibility of the corneas, the state of the pupils and extrinsic muscles, the acuteness and fields of vision and the state of the light sense are for the ophthalmologist to determine; the condition of the paranasal sinuses and of the auditory apparatus for the rhinologist and otologist; and the general examination of the nervous system for the neurologist. Wassermann tests should be made and in cases in which we can exclude tumor in the posterior fossa of the skull, spinal puncture and withdrawal of fluid is sometimes helpful in diagnosis and reduces intracranial pressure until operation can be done. The use of the spinal manometer, without withdrawal of fluid, will show whether there is increased pressure in the spinal fluid and hence increased intracranial pressure, thus helping one to differentiate between papilledema and papillitis.

A correct diagnosis is essential to treatment. When a diagnosis of papilledema is made, decompression will be

necessary, and medicinal treatment will be of no value except in cases of gummatous tumor. The most favorable cases as regards the preservation of vision are those in which symptoms other than visual make the patient submit to operation before deterioration of vision begins. In these cases, after successful decompression the papilledema subsides, the optic disc resumes nearly its normal appearance and vision remains unimpaired. When one waits until vision is affected, days or weeks will often pass before the patient's consent to operation is obtained, hospital arrangements made and new examinations carried out. By the time the operation is undertaken, usually central vision in one or both eyes is permanently impaired. One should early suggest operation contingently and have the surgeon examine the patient as soon as the obscurations of vision begin and before the permanent visual disturbances come on. Thus vision may be saved and we be spared the humiliating spectacle, so common today, of a patient sent for operation after useful vision has been irretrievably lost.

The most exhaustive treatise on papilledema is that by von Hippel, which appeared in 1921, in the second edition of the *Graefe-Saemisch Handbuch*.

THE APPLICATION OF IMMUNOLOGY TO OPHTHALMOLOGY.[1]

By Dr. ALAN C. WOODS, Baltimore, Md.

THE application of immunology to ophthalmology dates from the flood of experimental work on ocular anaphylaxis, which followed the early discoveries of Uhlenhuth and Arthus. In 1903, Uhlenhuth (1) discovered that the protein of the lens was apparently different from other body protein, that it was organ specific in its reactions. In the same year Arthus (2) discovered that localized anaphylactic phenomena could be produced. These two observations stimulated many investigators to inquire to what extent the eye might be subject to local anaphylactic phenomena, and what part ocular tissues might play in such a reaction. The great mass of experimental work thus stimulated was compiled in 1914 by von Szily (3). Although in many instances the evidence was conflicting, yet nevertheless certain broad principles were clear. Table I illustrates the broad principles underlying sensitization and anaphylactic intoxication of the tissues of the eye. Thus it is seen that through general sensitization of the body, that the ocular tissues are sensitized, and that if the intoxicating injection is given in the eye, local inflammation of the eye results, but if the intoxicating injection is given in the body, we have only symptoms of general anaphylactic shock. If the sensitizing injection is given in the eye, an especial sensitization of the eye results, in addition to a general sensitization. If the intoxicating injection be now given in the eye, local ocular inflammation is obtained, and little

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general reaction. If the intoxicating injection be given in the body, not only is a general anaphylactic shock obtained, but

TABLE I.

Sensitizing Injection	*Result*	*Intoxicating Injection*	*Result*
General (intra-peritoneal, intra-muscular, or subcutaneous).	General Sensitivity in which ocular tissues participate.	A. In ocular tissues. B. General.	Ocular inflammation. No ocular inflammation. General reaction only.
Local—in tissues of eye.	General sensitivity with especial sensitivity of ocular tissues.	General.	Ocular inflammation more marked than general reaction.
Local—in tissues of eye.	General sensitivity with especial sensitivity of ocular tissues.	Local—in tissues of eye.	Marked local ocular reaction. Little general reaction.

likewise an especial inflammatory reaction in the eye. From this it is seen that the eyes are sensitized as part of a general sensitization, the body tissues are affected by foreign protein absorbed from the eye, and, lastly, the eyes are capable of inflammatory reactions which are purely anaphylactic in character.

The second phase of the problem is centered in the effort to correlate these experimental studies to various diseases of the eye observed clinically. It is the purpose of this paper to briefly outline this phase, to point out ocular conditions which may have some relationship to the science of immunology, and very briefly to give the reasoning and experimental work on which such conceptions are based. The limits of time make it imperative that much important work be omitted from discussion. It is realized that in many instances the demonstration of the relationship of immune reactions to these ocular conditions is incomplete, and in other instances, scarcely commenced. The entire field is a most fascinating one for hypothesis and conjecture.

The various ocular conditions will be discussed in connection

with the ocular tissue to which they are preëminently related,—the conjunctiva, the cornea, the lens, and the uveal tract.

I. *The Conjunctiva.* The conjunctivitides which appear to be related to immune reactions are the conjunctivitides occurring in hay fever, hypersensitivity to pollens, food anaphylaxis, and phlyctenular disease.

The researches of Dunbar, Walker, Cooke, Coca, and others, have all indicated the dependance of ordinary hay fever on a hypersensitivity, either congenital or acquired, either to the pollen of certain plants or to animal epithelium. It has long been recognized that the mucous membrane of the eye, the conjunctiva, participates in the general irritability, hyperemia and congestion which affects the mucous membrane of the upper respiratory tract. Indeed, some observers have used the direct instillation of the pollen in the conjunctival sac, as a test to determine hypersensitivity to pollens, accepting the conjunctivitis that might result as a positive indication that the patient was sensitive to that especial pollen.

In the Spring and Summer months we continually meet a form of non-bacterial, irritative, conjunctivitis, which may vary from a mild conjunctival congestion, to follicular disturbances, and even up to typical vernal catarrh. The association of vernal catarrh with hay fever is recognized in the leading text books on ophthalmology (4). The recent advances in the knowledge of the etiology of hay fever has lead to much speculation if these peculiar conjunctivitides, and even vernal catarrh, may not be a symptom complex dependant upon the same mechanism as exists in hay fever. This idea has recently been emphasized by Townsend (5) who sums up much circumstantial evidence in support of such a view. The question is as yet too new to permit experimental research to be collected, but the fragmentary clinical studies available indicate that in certain seasonal conjunctival conditions a pollen hypersensitivity may be regularly found. During the past Spring and Summer, the author in collaboration with Dr. Leslie Gay, has studied several such cases of irritative conjunctivitis with folliculosis, and two cases of vernal catarrh, against the various pollens. The results of this study are shown in Table II. Six of the seven patients with conjunctivitis showed a pollen

hypersensitivity, and both of the patients with vernal catarrh showed a similar hypersensitivity. A. G. Fort (6) has also used

TABLE II.

SEASONAL CONJUNCTIVITIS × NEGATIVE BACTERIAL FLORA.

No.	*Clinical Condition*	*Result of Pollen Vaccination*	*Remarks*
1	Recurrent (summer) Follicular Conjunctivitis.	White Clover + +	Recurrence with clover pollenization. Normal recovery with cessation of pollenization.
2	Acute catarrhal Conjunctivitis.	White Clover + +	Clover in pollen at time of outbreak of conjunctivitis. Normal recovery with cessation of pollenization.
3	Acute follicular conjunctivitis.	Ragweed + +	Conjunctivitis showed period of exascerbation during period of ragweed pollenization.
4	Unilateral follicular conjunctivitis.	All Pollen tests Negative.	
5	Acute catarrhal conjunctivitis folliculosis, Hay fever.	Ragweed + + Other Pollens slightly positive.	Season recurrence of conjunctival symptoms in association with hay fever.
6	Recurrent kerato-conjunctivitis.	Red-top, Clover, Alfalfa. + +	
7	Vernal Catarrh.	Clover, Golden-rod. + +	
8	Vernal Catarrh (?)	White clover + +	Questionable diagnosis. Violent clinical condition. All bacteriological studies and animal inoculations negative.

this method of attack on the problem of vernal conjunctivitis. In his preliminary report, six patients with vernal conjunctivitis showed a positive reaction, in five instances, to Spring pollen and in four instances to Fall pollen. Fort went further and proceeded to desensitize the patients with the pollens to

which they were hypersensitive, a marked improvement resulting. Fort's experiences to date, given in a personal communication, are shown in Table III. This investigator has

TABLE III.

VERNAL CONJUNCTIVITIS.

(Studies by A. G. FORT, Atlanta, Ga.)

EOSINOPHILIA STUDY			POLLEN VACCINATION STUDY			TREATMENT BY POLLEN INOCULATION				
	Positive (over 4%)	*Negative (4% or under)*	*No. of Cases*	*Positive to Spring Pollen*	*Positive to Fall Pollen*	*With Spring Pollen (No. Treated)*	*With Fall Pollen (No. Treated)*	RESULTS		
								Cured	*Improved*	*Lost*
9	12	7	19	15	4	11			10	1
							4	1	3	

Spring Pollen: Timothy and Bermuda Group.
Fall Pollen:—Ragweed Group.

altogether studied twenty-eight patients with vernal conjunctivitis. In nineteen patients differential leucocyte counts were made, twelve patients showing a definite eosinophilia. Similarly, in nineteen patients, pollen vaccination tests were made. In each of these patients so tested, a definite pollen hypersensitivity was found. Fifteen of these patients were treated with pollen inoculations for the purpose of desensitization. One patient so treated was completely cured, thirteen showed definite improvement, and one patient was lost sight of. Such observations as these are most noteworthy, and strongly indicate the probability that vernal conjunctivitis is an allergic phenomenon dependent upon a pollen hypersensitivity.

Food Anaphylaxis. Conjunctivitis. Conlon (7) has called attention to the fact that conjunctivitis, unaccompanied by other manifestation may be secondary to a hypersensitivity to certain food stuffs, the hypersensitivity being probably congenital. He reported three such cases, one of recurrent conjunctivitis dependent upon dietetic indiscretions and brought on by indulgence in the specific food; the second case dependant upon a hypersensitivity to egg protein, and the last dependant upon an apparent hypersensitivity to certain fish protein. The last two cases both showed positive skin reactions to the suspected protein, and the condition disappeared

when the suspected food was withdrawn. Little is known of this condition, but in view of Conlon's experience it is worthy of mention.

Phlyctenular Kerato-Conjunctivitis. Notwithstanding the careful researches of such observers as Goldenburg (8), who associates the outbreak of phlyctenular disease with vagus irritability caused by faulty carbohydrate metabolism, and Turner (9), who associates the condition with disease of the ethmoids, it must be admitted that the evidence advanced in support of these views is presumptive, and that the weight of evidence, both clinical and experimental, indicates that phlyctenulosis is closely associated with a tuberculous infection. The clinical evidence in favor of such an association is, first, the well known incidence of the disease in individuals with a scrofulous or tuberculous "diathisis"; second, the high percentage of tuberculin hypersensitivity found in patients with the disease in 942 cases, collected from the literature by Gibson (10), in 1918, 801 showed a tuberculin hypersensitivity, (Table IV); and last, the definitely beneficial effects of tuberculin therapy. It is on these broad clinical observations

TABLE IV.

INCIDENCE OF TUBERCULIN HYPERSENSITIVITY IN PHLYCTENULOSIS. (AFTER GIBSON.)

Author	*No. of Cases*	*No. Positive*	*Percentage Positive*
Ayrenx	143	128	90
Bednarski	100	100	100
Belenky-Raskin	100	92	92
Bruns	102	62	61
Bywater	12	12	100
David and Vaugan	40	28	70
Derby	43	38	88
Pufer	49	41	84
Rosenhauch	50	48	96
Tivnen	50	46	92
Weekers[1]	156	142	91
Weekers[2]	58	32	55
Goldbach (added)	39	32	82
Total	942	801	85%

[1] Children. [2] Adults.

that the advocates of a tuberculous etiology for phlyctenules must, in the main, base their belief. On the other hand, the contentions, first, that the histological picture and clinical course of phlyctenules are not similar to that observed in tubercules; and second, that despite exhaustive research no one has yet been able to demonstrate tubercule bacilli in phlyctenules, have been urged against the tuberculous etiology of the disease.

The contention that the histological picture of a phlyctenule is not that of a tubercle need be given great weight. The structure of a phlyctenule,—a mass of epithelioid cells, in which lie a few polymorphonuclear leucocytes, surrounded by a zone of round cells of the lymphocyte type is certainly not dissimilar from the picture found in many types of tuberculous lesions. In fact the histological structure of a phlyctenule may be said to rather suggest a tuberculous process. The contention that tubercle bacilli are not present in phlyctenules must be given consideration, and it must be admitted that the weight of evidence is all against the presence of tubercle bacilli in phlyctenules. To explain how phlyctenules may be related to tubercular disease and yet not be caused by the tubercle bacillus *per se*, attention must be given to the possibility that they may be an allergic phenomenon related to a tuberculous infection. There is definite experimental and clinical evidence in support of such a view. This evidence may be somewhat clearer if the nature of the tuberculin reaction is considered.

In its broad principles, the tuberculin reaction is undoubtably an antibody-antigen reaction, as has been emphasized by von Pirquet (13), Wolff-Eisner (14), Wasserman and Bruck (15), and others. The researches of these investigators indicate, broadly, that tuberculo-protein substances, diffusing out from a tuberculous focus, cause the production of anti-bodies by the fixed cells, and that the tuberculin reaction represents the results of the union of such antibodies with the tuberculin. All tuberculins, if active, owe their potency to the specific protein of the tubercle bacillus (Hamman) (16). The more recent researches of Zinsser have explained some of the technical difference between a typical anaphlyactic reaction and the tuberculin reaction. Zinsser (17) believes that in the tuberculin reaction the antigen-antibody mechanism is involved, but that the process is probably transferred to the interior of

the cell, and the differences in the chemical and physical properties of the inciting agent explain the differences observed between the anaphylactic reaction and the tuberculin reaction. However this last may be, a positive tuberculin reaction may be definitely regarded as evidence of the sensitization of the cells of the body by the specific material of the tubercle bacillus, and the reaction itself definitely regarded as an allergic reaction dependant upon the sensitization of the cells. It is "an evidence of tuberculous infection, and not of tuberculous disease" (Hamman).

With this conception of the tuberculin reaction as an allergic reaction in mind, the direct evidence in favor of phlyctenules being an allergic phenomenon related to tuberculous infection may be considered. This evidence is quickly presented. First, Weekers (11), Rubert (12), Gibson (10), and other investigators have shown that in a certain percentage of tuberculous animals phlyctenules may be produced in the eye by the instillation of tuberculin in the conjunctival sac. Second, in patients hypersensitive to tuberculin, instillation of tuberculin in the conjunctival sac, as in the Calmette reaction, has caused the appearance of phlyctenules as part of the resulting positive tuberculin reaction. It is on these grounds, that both in experimental animals and in man, phlyctenules have been produced by the instillation of tuberculin in the conjunctival sac of the tuberculin hypersensitive, as part of a recognized allergic reaction, that phlyctenules may be regarded as an allergic phenomenon related to tuberculin hypersensitivity.

Extending this view to phlyctenules as observed clinically in the tuberculin hypersensitive, it seems necessary to assume that there must be an especial hypersensitivity of the ocular tissues to the tuberculo-protein. A most attractive hypothesis is to assume that in those individuals who suffer from phlyctenules and show a tuberculin hypersensitivity, there has been, at one time, a minute tuberculous focus in the eye which has produced an especial hypersensitivity of the ocular tissues. In such individuals, any further alteration of the tuberculin hypersensitive state, which may be associated with a liberation of tuberculous material, would readily cause a local tuberculin reaction in the especially sensitized eye, which might appear in the form of phlyctenules. This hypothesis is

strengthened by the following well known clinical observations. First, in the tuberculous, an alteration of the tuberculous condition, liberating tuberculous material, is accompanied by a well defined, general, tuberculin reaction. Second, if an individual who has previously shown a positive Calmette reaction be given a subcutaneous injection of tuberculin, the tested eye, even though entirely white when the subcutaneous injection is given, will react with an inflammatory reaction, while the second, untested eye will remain entirely quiet. This last observation indicates that an especially sensitized eye reacts especially to tuberculin, and is in accord with the experiments on ocular anaphylaxis previously mentioned.

If we accept this reasoning, and take the view that phlyctenules are an allergic reaction on the eye, dependant upon an especial sensitization of the ocular tissues, produced by further absorption of the specific material from a focus of infection, it does not necessarily mean that such focus of infection be tuberculous. The high incidence of tuberculin hypersensitivity in phlyctenular disease has already been mentioned and it would seem probable that tuberculo-protein be the material commonly responsible for the hypersensitivity and intoxication. However, a similar reasoning might well apply to other forms of focal infection, such as ethmoidal disease, assuming the eye has become hypersensitive either to the specific exciting agent or to the metabolic products of the infected focus, further absorption of such material producing an allergic reaction in the form of phlyctenules. It is, however, only in the instance of tuberculo-protein that we have the means of demonstrating the hypersensitivity, and producing clinical and experimental evidence in support of this view.

II. *The Cornea.* The experiments of Wessely (18), confirmed by other investigators show that a parenchymatous keratitis may be produced by experimental anaphylaxis. If one cornea of the experimental animal is sensitized by an intralamellar injection of the antigen, a later intoxicating injection of the same antigen, in the same, the fellow eye, or through the blood stream, produces a keratitis resembling closely, both clinically and histologically, the interstitial keratitis of lues. We have recently observed a keratitis in man which seems quite similar

in its etiology to this keratitis anaphylactica of Wessely. The patient, a negro farm hand, while working in a corn field in September of 1922, scratched the cornea of his right eye with a blade of corn. A keratitis rapidly developed which raged with increasing intensity for six months, in spite of all the usual local treatment. He was first seen in the clinic at the Johns Hopkins Hospital in March, 1923. At that time he showed a depressed furrow in the cornea, covered with epithelium and not staining. Surrounding this furrow were numerous plaques of interstitial, corneal, infiltration. The eye was intensely inflamed, and vision was reduced to light perception. A complete medical survey, including Wassermann and tuberculin reactions, was negative. Likewise all special examinations, bacteriological, laryngological, genito-urinary, etc., were negative. On vaccination with the various pollens and grains (by Dr. Leslie Gay) the patient was found to be intensely hypersensitive to corn. Treatment consisted in atropin locally, and desensitization with corn pollen. Early in the desensitization, as the dosage of corn pollen was increased, the eye showed a sharp exacerbation. The desensitization was then continued more slowly, and as the sensitivity was overcome, the eye became white, the plaques of interstitial infiltration disappeared, the furrow filled with scar tissue, and the central vision rose to $\frac{1}{1\frac{0}{0}}$. To our minds, it seemed that we were dealing with a keratitis anaphylactica, dependant upon the introduction of corn pollen in the cornea of a hypersensitive individual. An anaphylactic keratitis such as this is, of course, an accident in man and will probably be extremely rare. But that a similar mechanism may underlie many of the parenchymatous keratitides we observe clinically is not improbable.

The mechanism whereby the ordinary interstitial keratitis of syphilis occurs is far from clear. The clinical observations, first, that an interstitial keratitis may develop in an individual while under active anti-syphilitic treatment where all other indications indicate that the disease is under control and retrogressing favorably and second, that the interstitial keratitis usually runs its course, regardless of the most vigorous anti-syphilitic treatment, have long been a source of perplexity both to syphilographers and ophthalmologists. His-

tological studies and animal inoculation experiments have demonstrated that, as a rule, treponemata pallida are not present in the cornea in interstitial keratitis, although they may rarely so occur. On the basis of the experiments of Wessely, the theory has been advanced that the usual case of interstitial keratitis, excluding such exceptional instances as may be due to an actual invasion of the cornea by the treponemata pallida, is dependant upon a sensitization of the cornea by the treponemata pallida, or its products, during intra- or extra-uterine life. A further absorption of the syphilitic virus, during childhood or later life, in the sensitized cornea, produces a keratitis. Such a view has been advanced by Igersheimer, Derby, Walker and others. Recent, as yet unreported, experiments by Dr. Alan M. Chesney and myself, conducted in the Department of Experimental Syphilis at the Johns Hopkins University, have indicated that such an hypothesis may be correct.

These experiments seem to show that the cornea of a syphilitic animal may become sensitized to the syphilitic virus, toxin, or material, further absorption of such material producing an interstitial keratitis.

A similar reasoning may be applied to the interstitial keratitis observed in the tuberculous and the tuberculin hypersensitive, which lesions we are accustomed to class as tuberculous. Such keratitides do not present the appearance of tubercles, or simulate the type of pathological lesion caused by the tubercle bacillus. The significance of tuberculin hypersensitivity has already been referred to, and it may well be that many of the "tuberculous" kerititides are in fact an allergic phenomenon dependant upon a tuberculin hypersensitivity.

III. *The Crystalline Lens.* The relationship between immune reactions and lenticular conditions centers in the possible rôle lens protein may play in the causation of clinical conditions. Uhlenhuth's discovery has already been mentioned. This, and later researches along the same lines, have shown that the lens protein of various animals is alike, and that it is different from the other protein of the body—it is organ specific and not species specific. It has been demonstrated that an animal may be rendered sensitive to his own lens protein, and that

subsequent absorption of lens protein can produce allgeric phenomena.

Römer (19) was the first to attempt to attribute a clinical significance to this discovery. Römer's investigations, extending from 1905 to 1914, excited much controversy, and were not conclusive. In brief, Römer proceeded on the theory that senile cataract was the result of the action of some cell poison or cytotoxin, on the cells of the lens. The investigations covered a wide field:—the effect of lens protein on the blood of various animals (19), the liability of the lens to lipoids and to various poisons (20), the permeability of the lens capsule to toxins, serological studies in patients with cataract (21), and finally to specific organotherapy with lens protein (22). Although experiments by Lazarew (23) and other investigators failed to show a relationship between the immunological properties of lens protein and the formation of cataract, there are points in Römer's work which cannot be entirely ignored. Römer and Gebb were able to demonstrate, in the blood serum of a young diabetic with cataract, specific anti-bodies for lens protein. They were also able to show that the lens capsule was permeable for cytotoxic substances, and that the cells of the lens were capable of uniting with substances for which they had specific receptors. As a result of these studies, although Römer could not substantiate this theory by experimental research, he was convinced that the formation of cataract was due to a reaction taking place between the blood serum and the albumen of the lens (24). General as is this statement, it is not lightly to be dismissed, for the protein of the lens offers us the cleanest cut example of organ specificity we possess. It seems quite possible that in the life-long metabolism of the lens, this peculiar immunological property of lens protein may not remain entirely inert.

This entire idea has recently received great impetus from the brilliant experiments of Guyer and Smith (25, 26, 27). Although working on a different phase of the problem, these investigators took advantage of the fact that in embryonic life the lens is surrounded by a *tunica vasculosa* and is therefore more subject to influences from the blood serum than would occur in extra-uterine life. In their earlier experiments,

fowls were immunized to rabbits lens protein, producing anti-rabbit-lens serum. This serum was then injected back into pregnant rabbits, at two and three day intervals, over a period of about two weeks. There was a high mortality among these rabbits and the embryos, but among sixty-one young surviving, nine had ocular defects. The ocular defects were either in the crystalline lens—imperfect development, liquifaction of the lens, partial or complete cataract,—or, in the opinion of the authors, due to the effect of the imperfect development of the lens on the other ocular tissues. A similar experiment, using mice as the experimental animal, yielded the same results. In a still later experiment, in one instance, these same investigators were able to produce a similar lenticular lesion in the young by immunizing a pregnant rabbit directly with rabbits lens protein. These ocular defects, so produced, were hereditary in this strain of rabbits.

Guyer and Smith seem purposely to omit a discussion of the immunological phases of this experiment, apparently assuming that the lenticular defects were caused by the passive immunization of the pregnant rabbit with the anti-rabbit lens serum, a possible defect in the placental circulation allowing the maternal blood to come into direct contact with the foeti. However this last may be, which Guyer and Smith admit is a matter of conjecture, it is probable that the serological reactions are infinitely more complicated than they appear to assume. The introduction in rabbits of a fowl serum, especially repeated at regular intervals, as was done in this experiment, will most certainly result in the production in the pregnant rabbit of an anti-fowl serum. Now when the foreign serum is also an immune serum, as in this instance, it has been shown by Bordet (28) that the repeated inoculation in animals produces an anti-amboceptor (anti-sensitizer or anti-antibody) serum. Now Bordet has further shown that such anti-amboceptor receptors are not identical with those which anchor the amboceptor to the fixed cell, and are not specific for this amboceptor, but "neutralize unspecifically various unspecific sensitizers as well as normal antibodies in the serum of the same animal" (Zinsser). In the case of the startling experiments of Guyer and Smith, serological studies on the pregnant rabbits and their progeny are not available,

and any surmise of the possible mechanism underlying the production of the lenticular changes is therefore futile. The tremendous complexity of the problem is apparent. What factors of immunity and resistance may be involved, what diversion of a possible normal protective mechanism against lens protein may take place, and how far normal immune reactions may be vitiated, can scarcely be surmised. But when this experiment is considered, one is irresistibly reminded of the early statement of Römer:—that he was convinced that "cataract was due to a reaction between the blood serum and the albumen of the lens."

Verhoeff and Lemoine (29), in their excellent studies on endophthalmitis phacoanaphylactica, are the first, however, to demonstrate clearly that the remarkable immunological properties of lens protein, first discovered by Uhlenhuth, have at least one clinical relationship. It occurred to these investigators that the inflammatory reaction often observed following lens injury in certain individuals, might depend upon a hypersensitiveness to lens protein, either congenital or acquired, in these individuals. This hypothesis was made the subject of clinical, pathological, and experimental research. Verhoeff and Lemoine showed that in patients with rupture of the lens capsule, either traumatic, operative, or spontaneous, where intra-ocular inflammation occurred, a hypersensitiveness to lens protein could be demonstrated. In similar cases of rupture of the lens capsule, where uneventful healing occurred, there was no such hypersensitiveness to lens protein. Experimental work on guinea pigs confirmed these clinical observations. In guinea pigs, sensitized to lens protein, a discission of the lens capsule, which allowed the native lens to be absorbed as an intoxicating antigen, produced intra-ocular inflammation comparable histologically to the changes observed in human instances of intra-ocular inflammation following rupture of the lens capsule. This type of ocular inflammation the investigators aptly termed "endophthalmitis phacoanaphylactica." The characteristic histological picture consisted in the invasion of the lens substance by leucocytes. The clinical importance of this contribution is immense. By very simple skin tests an operator is able to determine the presence or absence of a hypersensitivity to

lens protein in a patient; and should the type of operation be such that lens substance may be freed for absorption, in the anterior chamber, the attempt can be made to de-sensitize the patient before operation, and thus avoid the outbreak of post-operative inflammation. Such a procedure has already been successfully carried out by Verhoeff and Lemoine (30). It is quite interesting that Verhoeff and Lemoine call attention to the similarity between endophthalmitis phacoanaphylactica, and serous traumatic iridocyclitis.

IV. *The Uveal Tract.* The principal disease involving the uveal tract, which has been brought into relationship to immunological phenomena, is sympathetic ophthalmia. In 1910, Elschnig (31) brought forward his theory of the anaphylactic nature of the disease, and in this year and the years following, presented certain experimental work to substantiate the theory. In brief, Elschnig believed that in sympathetic ophthalmia, the pigment epithelium, injured in the exciting eye, was absorbed from the eye by the patient. When this occurred, a condition of hypersensitiveness to pigment was created, especially in the fellow eye. Any further absorption of pigment now produced an anaphylactic intoxication, which when it occurred on the cells of the second eye, was manifested clinically as a sympathetic ophthalmia. Elschnig was able to show by his researches that the pigment of the uveal tract had organ specific properties, and was capable of acting as a foreign protein in the same animal. This problem has been the subject of further investigation by the author (32), who has been able to confirm, in the main, Elschnig's findings, and further to show (33) that in wounds of one eye, involving the uveal tract, where normal healing takes place and sympathetic ophthalmia does not result, circulating antibodies against pigment can be demonstrated in the blood serum of the injured person. In other words, normal healing is apparently associated with the development of an immunity against pigment. In cases where sympathetic ophthalmia develops following a wound of one uveal tract, this apparent immunity does not develop. On the other hand sympathetic disease appears to be associated with a definite cellular hypersensitiveness to pigment, which can be demonstrated by a skin

test. These findings, the development of pigment antibodies associated with normal healing, and the association of a hypersensitivity to pigment in sympathetic disease have been used as diagnostic aids to determine the status of a patient with injury to the uveal tract, where sympathetic disease may be feared (34). This will later be alluded to. Further, the author, in collaboration with Knapp (35) has made use of these findings in the treatment of sympathetic ophthalmia. This treatment is based on the conception of the removal of the factor we appear to find constantly associated with sympathetic disease, and the production of the factor we find associated with normal healing; in other words to remove the hypersensitivity, and to produce the circulating pigment antibodies. This has been attempted by the processes of desensitization and active immunization with uveal pigment. Such a procedure is extremely complex. Five patients have been so treated to date, and while in several cases the results were most encouraging, yet the complexity of the problem is realized, and a final opinion cannot yet be given.

Knapp (36), Guillery (37), and many other investigators, have called attention to the possible relationship of sympathetic disease to focal infection, tuberculosis, and other general conditions, removed from the eyes. This important point was first mentioned, rather broadly, by Elschnig (31), who called attention to the frequent association of sympathetic disease with other more general conditions. The discussion of this point is scarcely within the domain of this paper, but it may be remarked that the apparent difference between sympathetic ophthalmia on one hand, and normal healing of an ocular wound on the other hand, seems to lie in the difference between the development of a hypersensitivity and the development of a definite immunity. These may both be regarded as different stages in the development of the same immunologic process of the reaction of an organism to foreign protein. It is possible, although we have no experimental proof, that the presence of focal infection or systemic disease, may influence this reaction of the organism to uveal pigment and thus favor the development of a hypersensitivity, rather than allow the reaction to progress to the development of an immunity.

Idiopathic Uveal Disease. Experimental and clinical investigation have shown that the greater number of the commoner diseases of the uveal tract are probably of a bacterial origin. There remains a group, however, for which we can find no cause, be our medical studies ever so painstaking. The possible relationship of certain of these groups of idiopathic uveal disease to allergic phenomena is worthy of consideration.

There is a growing belief among students of internal medicine, for example, that certain forms of subacute and relapsing arthritis may in truth be allergic phenomena. This belief is strengthened by the demonstration that acute arthritic conditions may be produced by sensitization and intoxication by killed streptococci, and the clinical observation that relapsing serum sickness, which we recognize as an allergic condition, would in many instances be indistinguishable from some forms of relapsing subacute arthritis, did we not know the history. In such forms of arthritis, it may be supposed that from an old inflammatory process, definite damage has been done to the joint, and the tissues have become sensitized to the exciting agent, which need not of necessity be bacteria themselves, but may be the products of the metabolism, either of a bacterial or a non-bacterial inflammatory focus. Further absorption of such material, acting as an antigen or allergin, either through such a mechanism as von Pirquet supposed underlay serum sickness, or brought on by trauma, minor remote infections, gastro-intestinal absorption (food anaphylaxis), or other means, produces an exacerbation in the sensitized joint, which may be interpreted as an allergic phenomenon.

Such an hypothesis as this may well be extended to ophthalmology as a possible explanation for certain of the idiopathic uveal diseases we encounter. Especially may it apply to those types of uveal disease we have been long accustomed to associate loosely with "rheumatism." It may well explain certain of the conditions we are accustomed to ascribe to as a "toxæmia" from a known source of infection, for it is vastly more reasonable to suppose that sensitization and intoxication may occur through the lymphatics or blood stream from the metabolic products of a given focus of infection, than to suppose a hypothetical toxin, the existence of which we

have neither clinical, chemical, or experimental proof. It must be remembered, of course, that we have no experimental support for such an hypothesis, but as a clinical suggestion, especially in the light of modern medical thought, it is worthy of consideration.

Methods of Diagnosis.

There are various diagnostic procedures which may be used to demonstrate the immunological reaction underlying a pathological process. The application of such methods to the practice of ophthalmology represents the practical application of the science of immunology to the study of diseases of the eye. The principle pathological conditions of the eye which may be related to immunology have already been mentioned. It may be well, in conclusion, to briefly mention the procedures which are available to demonstrate such a relationship clinically.

1. *Pollen Sensitivity*. The best preparations of pollens for these tests are those prepared by the methods described by Coca. The test consists in the skin reaction observed when the pollen to which the patient is hypersensitive is applied to the skin. There are two methods of making the skin test, the cutaneous, or scratch method, and the intradermal method. Brown (39), who has made a study of the relative value of these two methods, believes the intradermal method to be better, less painful and giving more accurate results. In making these tests, it is important that the patient be tested against the individual pollens, especially those prevalent at the time of the outbreak of the disease. The use of mixed pollens, mixtures of Spring and Fall pollens, is scarcely to be recommended since more accurate diagnosis is possible, and should desensitization be considered, it is better to determine the individual pollen responsible for the hypersensitivity, so the desensitization may be specific.

2. *Tuberculin Hypersensitivity*. The methods of determining tuberculin hypersensitivity are based on the reaction observed when small amounts of tuberculin are introduced into the body. These reactions consist in the local reaction (the reaction observed around the point at which the tuberculin has been introduced), the general reaction (fever, etc.), and

the focal reaction (the lighting up of a tuberculous focus). The usual methods of introducing tuberculin are the (1) subcutaneous test, (2) the cutaneous, or von Pirquet test, (3) the conjunctival or Calmette test, and (4) the intracutaneous test.

In ophthalmological conditions, the most important thing to guard against is the occurrence of a focal reaction in the eye. For while this will leave little doubt of the diagnosis, it may well defeat our ends, and destroy the eye we are attempting to save. Focal reactions are probably observed with greater frequency in the subcutaneous test than in any other, and for that reason, this test should be used most guardedly in ophthalmology. Except for this danger, this is perhaps the most valuable of the several tests. The von Pirquet test is of little value, except in children, on account of its great delicacy. The Calmette test is not to be recommended, the danger of a combined local and focal reaction make it totally inapplicable to ophthalmological conditions. In the experience of the author, the intracutaneous test is the most valuable. In this test, varying dilutions of tuberculin are used, starting at 1:100,000, and increasing the strength to 1:100. Several injections can be made at one time, thus 1:100,000; 1:10,000, and 1:1,000, dilutions may be injected the first time, with proper controls. The injection is made into the layers of the skin, $\frac{1}{10}$ *cc* being injected. If the patient does not react to these injections, stronger dilutions may now be used. In this manner, we not only determine whether the patients may be hypersensitive to tuberculin, but also determine the degree of hypersensitivity, a determination which is of great value if tuberculin therapy is to be undertaken, besides the possible clinical value of such a determination. Further, this method of using tuberculin appears to be entirely safe. Focal and general reactions do not occur. In the opinion of the author, it makes little difference which preparation of tuberculin is employed, provided it is a tuberculin of known potency.

Wassermann Reaction. There is but one point concerning this well-known diagnostic procedure, which seems worthy of mention here. A negative blood serum reaction, even after a provocative arsphenamin, is not to be relied on as excluding the diagnosis of active syphilis, if the case under considera-

tion is an optic neuropathy. In a series of optic neuropathies recently studied at the Johns Hopkins Hospital, of twenty-four patients with primary atrophy of the optic nerves, presenting definite serological evidence of syphilis, five patients had negative blood serum Wassermanns, while the spinal fluid was strongly positive. Experience with neuro-syphilis in other clinics amply bears out this observation.

Lens Protein Hypersensitivity. In detecting hypersensitivity to lens protein, Verhoeff and Lemoine make use of the usual skin tests, the cutaneous and the intradermal tests. The lens protein is readily prepared. Pigs' lenses are removed under sterile precautions, dried, and ground to a fine powder. For the cutaneous tests, the powder itself is used, while for the intradermal a 10% solution by weight in normal saline is used. The technique of the tests is generally the same as when these methods are used for testing with tuberculin. In the cutaneous reaction, the local hyperæmia and elevation, which denote a positive reaction, become evident within an hour. In the intradermal reaction the same phenomena are noted from two to twenty-four hours after the test.

Pigment "Immunity" and Hypersensitivity Tests. These tests have been employed by the author, and appear to give knowledge concerning the status of a case as regards sympathetic ophthalmia. The pigment antibodies are detected by a complement fixation test, using the pigment suspension as the antigen. This reaction is quite delicate, and the reagents require constant titration that positive reactions may be detected. Further, the antigen has a tendency to become rapidly anticomplementary, and must be frequently renewed. For these reasons, the author does not believe that this test will have much practical value except in a research laboratory, where it is kept in constant use. The pigment hypersensitivity test, however, is free from technical difficulties, is quite easy, and appears capable of general application. This test seems to be the more valuable of the two from the standpoint of the clinician, for it tells whether the patient is hypersensitive to pigment, which is apparently the vital point in relationship to sympathetic ophthalmia. For this test, three dilutions of pigment are usually employed: a normal suspension, a 1:10 dilution, and a 1:50 or a 1:100 dilution. Intradermal

injections are made on the flexor surface of the forearm, each about the size of a dime. As a control a similar injection is made on the other forearm, using normal saline, to which 0.15% tricresol has been added. A positive reaction consists in local hyperæmia and elevation around the site of the injections coming on in from one to twenty-four hours. Occasionally, more remote general symptoms have been observed. Such symptoms are slight fever (1–2°), slight malaise or asthmatic symptoms, and slight generalized urticaria. Frequently, urticaria may be observed around the site of the injections.

COMMENT.

It must be clearly understood that the greater number of the ideas advanced in this survey are far from proven. Whether they are capable of conclusive proof is an open question. Certainly such proof will, in the greater number of instances, not be forthcoming for years, and even then may be only clinical proof. The chapter of immunology in the history of medicine is far from finished. In many respects we still stand on the threshold, explanations which yesterday were accepted are today no longer tenable in the light of more recent research. But more and more we realize, as our knowledge advances, the enormous rôle that allergy occupies in the progress of disease. The recent advance in our knowledge of syphilis and tuberculosis amply illustrate this. Early pathological researches in ophthalmology pointed out the difference between the acute type of lesions produced experimentally by the sensitization and intoxication, and the chronic type of lesions found in man in various clinical diseases to which an analogy had been drawn. Such differences in pathology were assumed by these investigators to be proof of a different etiology. Such a contention no longer holds, for later in vestigators, notably Longcope, have been able to produce a variety of chronic degenerative lesions by injections of foreign protein.

The application of immunology to ophthalmology even in the present state of our knowledge, offers us an explanation for certain ocular conditions, which are not now explicable upon any other basis. Whether these explanations will prove to be true only time and further observation and experimentation will show.

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CONCERNING SOME GROSS STRUCTURAL ANOMALIES OF THE MUSCLES OF THE EYE AND ITS ADNEXA.[1]

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(With five illustrations in the text.)

FUNCTIONAL disturbances of the eye muscles are common, and for the most part easy of recognition by the ophthalmologist; those the subject of this paper, as embodied in the title, are rare, though in all probability occurring much more frequently than is usually supposed.

Positive evidence of the existence, position and structure of any one of the muscles moving the eye or the lids can be obtained only by actually viewing the muscle in question, which naturally is alone possible antemortem at operations, or postmortem by dissection.

In many cases of congenital squint, the supposition of the absence of a muscle arises when there is a total absence of motion of the eyeball in the field of action of such muscle, but such observations are purely hypothetic, being without definite basis. Even at the time of operation, it is impossible to reach any satisfactory conclusion regarding the condition present, for, owing to the conicity of the orbit, the surgeon can see, in the case of the muscles attached to the eye at least, but the proximal end of the muscle engaged. All estimation as to the extent, origin, course, etc., of the remainder of the muscle must be conjectural.

Until the present at least, postmortem dissections of the orbit have been comparatively few. The average undergraduate medical student gives but scant attention to that

[1] Read before Am. Acad. Ophth. Oto.-Lar., Washington, Oct., 1923.

part of the cadaver, and research in that field has been left pretty much to the anatomists, and apparently but few of these have devoted much time to orbital dissection.

Just as ophthalmic surgeons, however, with large experience have not infrequently encouraged marked structural anomalies of the muscles in their squint operations, often as a source of considerable embarrassment at the time and greatly interfering later with the results they had desired to obtain, so the few anatomists who have dissected many orbits found, not infrequently, marked anomalies in the musculature of the eyes and lids. In his excellent work on the anatomy of the orbit, Whitnall acknowledges this in the following words: "It is probable, to judge from the writer's individual experience in finding quite a number of gross abnormalities of the ocular muscles in his series of dissections, that such are by no means as excessively rare as would appear from the number recorded in the literature; dissecting room conditions do not favor their identification, and in life some may be unrecognizable through compensatory action of the other muscles."

In describing the few cases of his own which the writer desires to record, it may not be amiss to interpolate a synopsis of the literature, and in so doing to consider the subject under two headings, including (1) supernumerary and anomalous muscles of the lids and orbit, and (2) those of the eyeball itself. Concerning the former, one cannot do better than quote from Lucien Howe's classic work on Muscles, as follows:

"SUPERNUMERARY MUSCLES OF THE ORBIT. So little mention is ordinarily made of the supernumerary muscles, that one not accustomed to dissections may be surprised perhaps that such muscles exist. But anyone who has observed the variations in the secondary insertions in different individuals, or who knows how the bands of connective tissue in different parts of the orbit vary in their distribution, can easily understand how these bands may be sufficiently marked in certain cases to be described as special muscles.

"For it must be understood that none of these supernumerary muscles approaches in size the other extrinsic muscles, but consist only of bundles of striped muscular fibers interwoven with connective tissue fibers.

"1. *Muscle of Horner.* Among these supernumerary muscles,

attention should first be called to the muscle of Horner. This is seldom mentioned in descriptions of the contents of the orbit, yet it is often of considerable importance in connection with the operation for tenotomy of the internal rectus. * * *

"2. *The Gracillimus or Transversus.* At quite an early date, careful anatomists recognized at least one or two other supernumerary muscles—these lying near the roof of the orbit. Albinus found a band passing from the levator with connections inward, especially to the superior oblique, and this muscle he called the gracillimus. * * *"

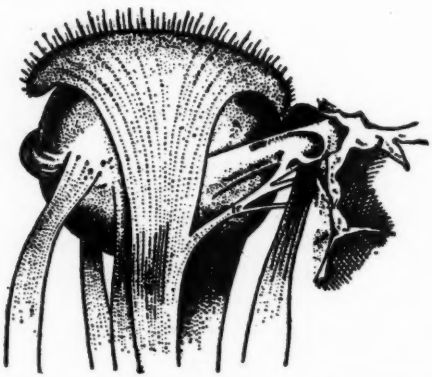

FIG. 1. Supernumerary muscle, the transversus.

A more careful search for the gracillimus, or a muscle similar to it, was made by Budge. He says: "While making preparations of the muscles of the eye, I found a muscular portion given off from the levator palpebræ superioris. This portion branched into two small bundles from the inner side of the muscle, and then, passing inward, they were inserted into the trochlearis." He examined about thirty orbits of children and grown persons as to this point, and there were only five without some traces of these fibers, although in some instances they appeared to be hardly more than threads.

Bochdalek describes another as the "anomalous transversus." (Fig. 1) arising from the anterior and upper portion of the orbital plate of the ethmoid, it passes almost directly across the upper part of the orbit. At its origin, it consists of

small tendinous bands, 3 to 4*mm* in thickness; these enlarging into fleshy bundles, give off various attachments to neighboring facia, and especially to the levator palpebræ.

In a recent dissection, Rush and Schaeffer found a slender but well-defined supernumerary muscle, Fig. 2, which they thought was in some respects in agreement with the muscle described by Budge, the insertion of the muscle being, however, more extensive. Their description is as follows: "It is a well-defined muscle of from 2 to 3*mm* in thickness, which had a common origin with the levator palpebræ superioris along

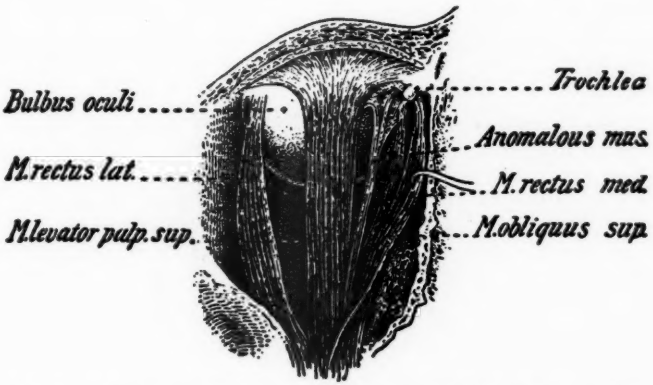

FIG. 2. A drawing of an actual dissection of some of the extrinsic muscles of the eyeball, showing, in addition to the usual muscles, the origin, insertion, and relations of the supernumerary or anomalous muscle.

its medial border. At a distance of 12*mm* from their joint origin, it parted company from the levator, and passing forward, it assumed a position in the orbit almost equidistant from the obliquus superior, the rectus medialis, and the levator palpebræ superioris. At a distance of 30*mm* from its origin, the muscle spread out into a peculiar fan shaped attachment. The stronger central fibers of the fan extended forward in two or three strands to be attached to the upper and lower borders of the trochlea, and even to the frontal bone beyond. A lateral expansion of the fan was directly continuous with a similar expansion from the levator, and an inferior expansion

was attached to the superior border of the rectus medialis. It received nerve filaments from the branch of the oculomotor nerve which supplied the levator. Traction upon the muscle gave a feeble internal movement of the eyeball."

In inquiring into the causes of congenital ptosis, Briggs noted the following underlying conditions at fault.

"(a) Defective development of levator and other muscles of the eye; (b) Adhesion of muscles; (c) Abnormal insertion of muscles; (d) Connective tissue bands instead of muscles; and finally (e) Absence of muscles."

Anomalies of the Musculature of the Eyeball Itself. The literature contains a number of accounts of anomalies of muscles governing the movements of the globe itself—some quite complete, others more or less fragmentary. The majority relate to the absence of a muscle. Thus, Krause, Ledouble and Lawford noted absence of the internus; Krause and Bahr of the externus; Seiler and Steinheim of the superior rectus; Stieren and W. E. Davis of both inferior recti; Coover of the superior and inferior recti; Hartes and Seiler of the obliques. Absence of all the eye muscles is described by Klincosch.

In operations for the correction of congenital strabismus, the writer, in not a few instances in his own cases and those of his colleagues, has noted an apparent complete absence of the rectus muscles, in other instances much attenuated muscles, and in still others the replacement of the muscle by a fibrous band. Thus, in one case of defective downward motion in the right eye recently operated on by him, he was unable to find a trace of the inferior rectus muscle. The anomaly occurred in a young man, the left side of whose face was underdeveloped; with the left eye fixing horizontally, the right eye deviated strongly upwards. The right eye could not be moved down and out, although it could be turned by the superior oblique somewhat down and in. At operation, the inferior rectus was found to be absent, except a very rudimentary portion at the usual site for its insertion. To this, the lower halves of the internal and external tendons were sutured, and a free tenotomy done on the superior rectus, care being taken to bring forward as much as possible the lower part of the capsule of Tenon. At the end of two weeks, the eyes were directed

on the same plane and there was a left hyperphoria of 10°. (*A. J. O.*, 1921, v. 4, p. 524.)

Muscles merged together or with union of neighboring parts have been reported by Olbers and Wrisberg. Morgagni recorded a union of the superior oblique with the trochlea, while Macalister found the fascia which often binds the rectus internus and inferior at their origin, to be extended, so that both these muscles were united from the posterior third of the orbit. The external rectus may be inserted into the globe by two tendons, as found by the writer, in the eye of a boy during a squint operation. (Fig. 3.)

The right eye had converged from birth and glasses, though worn since six years of age, had not corrected the deformity. Examination showed an alternating concomitant converging squint of 45°. A double advancement was performed, at which

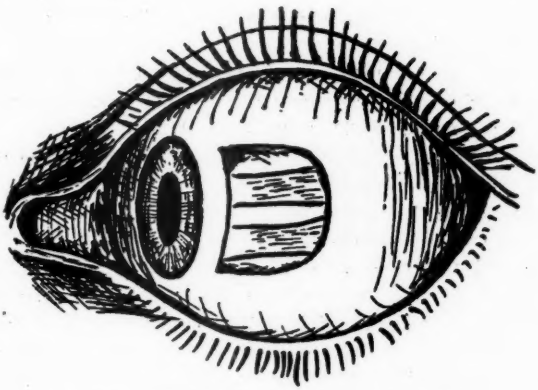

FIG. 3. Bifurcation of external rectus muscle.

time, in place of the single insertion of the left externus, the muscle was found to be inserted into the globe by two tendinous bands, symmetric in size and development, and about 4*mm* apart. The tendons blended into two well-developed muscular bellies, which ran parallel to one another until they were lost posteriorly in the tissues of the orbit. Whether they continued as separate muscles to the apex of the orbit, or formed a common belly before arriving at this position, was

of course impossible of ascertainment. The right externus was inserted normally. The results of operation by the usual twin mattress sutures were satisfactory.

This case is somewhat analogous to Wicherkiewicz's, who found while performing an advancement of the internal rectus, that the muscle was inserted by two tendons, separated vertically from each other by a distance of *4mm.* Dissection revealed that this space decreased posteriorly so as to form a triangle, with its apex in the belly of the muscle, *16mm* from the line of insertion. Macalister, whose treatise on anomalies of the ocular muscles is one of the most interesting on the subject, observed the external rectus supplied with two heads. Dieffenbach and Behr also noted instances of bifurcated muscles, while Zagorsky and Albinus found double external recti muscles.

A lateral rectus with two extra fasciculi, which passed forwards to terminate in the inferior tarsal plate and lateral wall, is recorded by Curnow.

In one of his specimens, Whitnall found a well-marked fleshy bundle, *7mm* long and *2mm* in diameter, passing from the lateral rectus across the posterior third of the orbit, beneath the optic nerve, to fuse with the belly of the medial rectus; no nerve could be traced to it.

Gross deviations in the location of the insertion of the tendons have been recorded by Rossi, Dieffenbach, Pflüger and others. Ter Aruntinjanz, at an operation for strabismus, found the internus tendon very broad and adherent to the sclera by an insertion above the normal one. The superior rectus was very strongly developed, while the inferior rectus consisted only of a feeble tendinous bundle.

In advancing the left external rectus, Bourgeois found no trace of it at its usual insertion, but discovered it inserted far superiorly and in oblique direction. The muscle was much atrophied, scarcely *2mm* broad.

In a case of retraction of the eye, Türck noted absence of the rectus externus. The internus was inserted *12mm* from the corneal limbus, and by a second head still more posteriorly, which accounted for the retraction.

The superior rectus was found by Aubaret to give off a muscular slip *15mm* long, which arose from the same origin from

the annulus of Zinn and passed downwards and forwards across the lateral face of the optic nerve to join the inferior rectus about its midpoint; the nerve supply came from the inferior division of the third nerve.

In a preparation studied by Whitnall, the inferior rectus could be seen giving off a large muscular bundle, which passed lateral to the optic nerve and joined the superior rectus; it was innervated by the lower division of the third nerve.

In his text book quoted above, Whitnall states that "the superior oblique may be closely accompanied by an offshoot from the levator palpebræ superioris, sometimes called the "comes obliqui superioris."

The inferior oblique was found by Whitnall to have an abnormally placed origin in several instances. Out of 100 orbits examined by him, the origin was in the usually described position immediately adjacent to the incisura lacrimalis of the maxilla

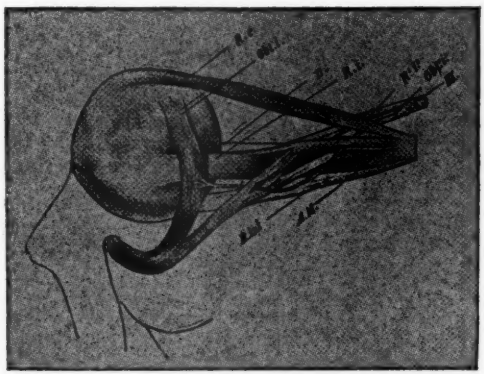

FIG. 4. An accessory inferior oblique muscle.

in 45 instances; it lay from 2 to 5*mm* distant in 47 cases (most often in the left orbit), and was situated from 6 to 7*mm* lateral to the normal position in 8 cases.

Rex described an abnormal muscle bundle (musculus obliquus accessorius inferior) binocular, which passed from the apex of the orbit to the inferior oblique, sending a slip to join the inferior rectus; both were supplied by the third nerve.

(Fig. 4.) This abnormal muscle of slender proportions had an origin from the orbital apex close to that of the rectus inferior and externus which blended with the muscle belly of the former and accompanied it to the interior portion of the orbit. Its insertion with the normally sized and inferiorly situated oblique was through the medium of a broad fleshy band of muscle fibers, about *6mm* from the point of origin of that muscle.

In correcting a case of right convergent squint of high degree in a young man, which had dated from birth, upon exposure of the tendon of the external rectus muscle, the writer discovered an accessory muscle just inferior to it. This muscle, which was inserted into the globe by a short, rounded tendon, about 4 to *5mm* long, immediately below that of the externus, was well developed, more or less rounded, and took an oblique

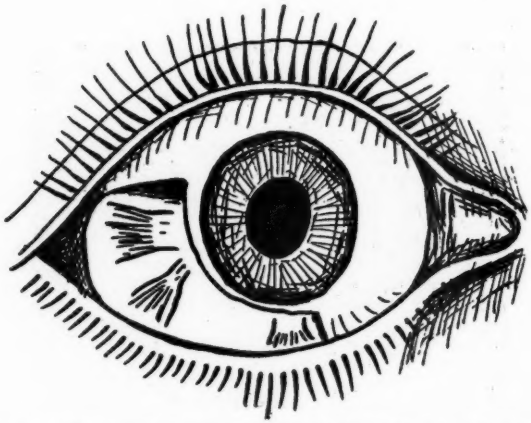

FIG. 5. An anomalous muscle, in association with external rectus.

course inferiorly, coursing between the externus and inferior rectus muscles, until lost in the tissues at the equator of the globe. Both the external rectus, which was somewhat attenuated, and the anomalous muscle were freed from their insertion and advanced by two twin mattress sutures. The result of the operation was excellent, parallelism of the visual axes being obtained. (Fig. 5.)

In a case of convergent strabismus observed by Baumgarten, the muscle itself was normal but enveloped in a dense connective tissue. Uhthoff saw, in place of a rectus internus, a normally inserted band of fully developed connective tissue. In a case which showed complete left lack of abduction, Bernheimer found a thin band of connective tissue in place of the externus. Axenfeld reported a similar case in a young man, who was unable to abduct the eyes to the left, although there was binocular vision in looking straight ahead and in the right field of vision. Other instances of replacement of the externus by a more or less inelastic cord have been reported by Baumgarten, Inouye, Alling, Schürenberg and Evans.

Vestiges of the musculus retractor bulbi (retractor oculi, superior oculi, rectus posterior, choanoid) muscle have on rare occasions been recorded in man, and what would appear to be a well-marked example was recorded by Whitnall.

Other instances have been noted by Nussbaum, and Fleischer (1907), and according to Lewitsky (1910) it is always represented in man (thirty specimens examined) by a well-marked strand of connective tissue (fascia retrobulbaris), lying between the lateral rectus and the optic nerve, and attached to the back of the fascia bulbi anteriorly; it does not appear, however, to be readily indentifiable.

According to anatomists, all these anomalies which have been cited are to be explained by intrauterine errors in development, by cleavage from the common premuscular embryonic mesoblastic mass.

CONCLUSIONS.

The foregoing is a fairly complete record of the gross anomalies of the muscles of the eye and its adnexa contained in the literature, and in the opinion of the writer possesses more than an historic interest, and is worthy of more than a hurried survey. All must have been accompanied by deviations in the ocular axes which were aberrant and confusing to an examining clinician, and perplexing and baffling to an operator desirous of straightening the eyes. Many cases of congenital squint are dependant upon such anomalies, and it is only by an appreciation of this fact and a better knowledge of these peculiarities, that the surgeon by modifying his plans can over-

come the difficulties in operating they entail and can succeed in establishing parallelism of the visual axes.

More frequent and more careful dissections of the human orbit are necessary. Ophthalmologists should familiarize themselves, also, with the orbital anatomy at least of the lower animals, for many of the anomalies enumerated will be found to be but reversions to lower types and not purely of adventitious occurrence.

MELANOTIC EPIBULBAR TUMOR DISPELLED BY THE USE OF RADIUM.[1]

By WILLIAM H. WILDER, M.D.
CHICAGO.

(With four illustrations on Text-Plate XVI.)

THE following case of a densely pigmented growth of the sclera, involving the limbus of the cornea, presents in its course and in its response to the action of radium, such interesting features that it seems worthy of record.

Miss L. C., age 41 years, first consulted me February 3, 1920. She was a healthy, well-formed white woman, 5 feet, 1½ inches in height weighing 110 pounds, a distinct brunette with black hair, dark brown irides, and clear healthy complexion.

She had measles, whooping cough and mumps in early life and at the age of 7 years scarlatina, and one year later typhoid. She suffered in early life from constipation and supposed liver trouble. Menstruation did not begin until the age of 17 years and then was rather irregular and more profuse than normal. When she was 25 years old she began to suffer with attacks from, what was called, chronic appendicitis, and later suffered with sciatica for several months. At the age of 34 years an appendectomy was done, and a small cyst removed from one ovary, after which she had no more sciatic attacks, and menstruation became normal and regular. She states that her skin was darker and more sallow before the appendectomy than it has been since the operation. For some time before the appendectomy, and up to a time about three years before I first saw her, she suffered at intervals from attacks that were called by

[1] Read before the American Ophthalmological Society, Colorado Springs, June, 1923.

her doctor "gall-bladder disturbance," causing nausea and pain in the region of the liver, although she never became jaundiced. The gall bladder had never been operated on, and she has had no attacks in the last six years. She has no moles nor pigmented spots on the body or limbs.

Family History. Father lived to the age of 70 years, had always been healthy, as were also her brothers and sisters. Mother's father lived to be old. Mother's mother died at age of 25 years from infection following child-birth. Patient said there was no history of cancer in the family so far as she knew.

Since early childhood there had been noticed a small pigmented spot on the temporal side of the eyeball not far from the cornea. About four years before the time when she first consulted me, she thought this spot was slowly increasing in size, and in two or three years more, she was sure it showed signs of active growth. She sought treatment from a local specialist who used drops and ointments for six months without effect. Later she consulted other specialists in towns near her home in Illinois, who thought the growing mass could be dissolved with drops, and one who advised its removal with an electric cautery, which, however, was not done.

A short time before I saw her, she had consulted Dr. J. Lifschutz of Chicago, who agreed that the growth was malignant and advised that the eyeball should be enucleated. As the eye had good vision, caused her no discomfort, and seemed to her to be in every other respect normal, she declined to accept the advice given her, hoping that some other treatment might benefit.

Examination showed a flat mass on the temporal side of the left eyeball, irregular in outline, about 10*mm* in its vertical dimension, about 8*mm* in its horizontal dimension, heaped up around the temporal limbus and pushing over the cornea about 1*mm*. It seemed to vary in thickness from 1 to 2*mm*, being somewhat thicker near the limbus and thinner as it spread over the sclera. It was deeply pigmented in places and there was considerable pigment in the episcleral tissue around it. The edge that was advancing over the corneal margin was distinctly demarcated and was light gray in appearance. Numerous small blood vessels could be seen in the tumor with a loupe and it was supplied by several larger vessels that coursed over the temporal side of the eyeball and in the conjunctiva.

Some idea of the appearance can be gained by the photograph (Fig. 1), which, however, lacks the coloration and does not show the height of the mass.

ILLUSTRATING DR. WILLIAM H. WILDER'S ARTICLE "MELANOTIC EPIBULBAR TUMOR DISPELLED BY THE USE OF RADIUM."

FIG. 1.—Appearance of epibulbar tumor, February 10, 1920 (before treatment).

FIG. 2. — Appearance, May, 1922, showing recurrence after the growth had largely disappeared following 86 treatments, aggregating 1132 mg. hours with emanation tubes.

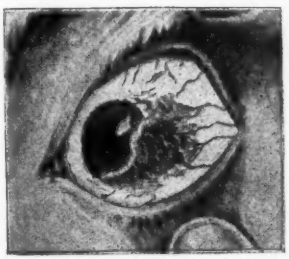

FIG. 3.—Appearance, December 1, 1922, six weeks after the conclusion of the last series of nine treatments, from May 8, 1922, to October 10, 1922, with radium plaques and tubes aggregating 110 mg. hours. Tumor has disappeared. Scattered areas of pigment still present.

FIG. 4.—Appearance, May 15, 1923. Growth has disappeared, as well as most of the pigment. No treatment since October 10, 1922.

There was no sign of any involvement of the iris or ciliary body. The pupil reacted normally to light and accommodation, and dilated evenly and widely after instillation of homatropin solution 2%.

Refraction under cycloplegic showed:

R.V. with Sph. + 2.00, Cyl. + 0.50, 75° = $\frac{20}{20}$.
L.V. " " + 1.50, " + 0.25, 15° = $\frac{20}{20}$.

The media were perfectly clear and the eye grounds appeared normal. Transillumination gave no indication of a growth in the interior of the eye.

The tumor presented all the appearances of a melanotic sarcoma, and the patient was informed of the risk she ran in attempting to save the eye. As she suffered no discomfort from it, and had as good vision as she had ever had in that eye, she refused to consider enucleation, and hence treatment with radium was suggested. There was no clinical history of lues, and Wassermann tests were negative.

Treatment was begun and carried on in great part in the Frank E. Simpson Radium Institute of Chicago, and later in the laboratory of the Physicians' Radium Association of Chicago. The treatments were carried on by emanation tubes of 50*mc* screened with 2*mm* of gold held at a distance of 4*mm* from the growth for 15 minutes. The healthy cornea and the rest of the eyeball was protected by a thick gold screen fashioned in the shape of an artificial eye. The following shows the number, duration, dates, amount of radium used, screening, distance, and millicurie hours of each series of treatments:

reat.	*Duration each treat.*	*Dates*	*Amt.*	*Total hrs.*	*Screen*	*Dist.*	*No. hrs.*
		1920					
10	15*min.*	Feb. 3, 4, 5, 6, 7, 9, 10, 11, 12, 13	50*mc*	2½	2mm gold	4mm	125
10	" "	Mar. 2, 3, 4, 5, 6, 8, 9, 10, 11, 13	50*mc*	2½	" "	"	125
10	" "	Apr. 5, 6, 7, 8, 13, 14, 15, 16, 17, 23	50*mc*	2½	" "	"	125
10	" "	May 21, 22, 24, 25, 26, 27, 29, June 2, 3, 4	50*mc*	2¾	" "	"	137
6	" "	June 25, 26, 28, 30, July 1, 2	50*mc*	1½	" "	"	63
7	" "	Sept. 2, 3, 4, 10, 11, 13, 14	50*mc*	1¾	" "	"	87
8	" "	Oct. 26, 27, 28, 29, 30, Nov. 1, 2, 3	50*mc*	2	" "	"	100
		1921					
11	" "	Mar. 26, 28, 29, 30, Apr. 1, 4, 5, 8					
	" "	11, 13, 15	50*mc*	2¾	" "	"	137
6	" "	June 8, 9, 10, 11, 13, 14	50*mc*	1½	" "	"	75
2	" "	Aug. 24, 25	125*mc*	½	" "	"	63
3	5 "	Nov. 4, 7, 9	125*mc*	¼	" "	"	32
		1922					
2	15 "	Jan. 19, 21	125*mc*	½	" "	"	63
86							Total 1132

Up to July 2, 1920, the patient had received 47 treatments in 5 series with intervals of rest. It was evident that the growth was much smaller and in places had almost disappeared. Pigment was scattered rather widely over the sclera beyond the site of the fleshy growth. She received another series of treatments in September, October, and November, 15 in all, after which treatment was discontinued for a time. It was noted that the fleshy growth on the sclera had almost entirely disappeared, but there still remained a slightly elevated membrane-like growth along the temporal limbus of the cornea. There were rather dense irregular pigment patches in the conjunctiva and in the episcleral tissue distributed widely above and below the original site of the tumor.

The patient was not seen by me for several months, having gone to her home in the central part of Illinois, but on March 26, 1921, when she came again, it was noted that the growth seemed to have become active and seemed to have spread along the lower margin of the cornea. The pigment patches were still pronounced in the tissues above and below the growth.

She was again subjected to the same treatment as before, receiving eleven applications of rays of fifteen minutes duration from a 50*mc* emanation tube at intervals during the next twenty days. An interval of rest of two and a half months followed when no treatments were given.

In the second week of June, 1921, she again received six daily treatments of the same dosage and duration, and it seemed as if the growth was being checked. On July 14, 1921, Dr. George de Schweinitz saw the case in my office and agreed with me that it had all the appearance of a melanotic sarcoma and that enucleation would be the best procedure. He suggested that a few large doses of radium should be given instead of repeated smaller doses.

Two treatments, 15 minutes duration, with 125*mc* were given on two successive days in August, followed by a period of two months rest. It seemed as if the growth had been again arrested.

In November she received three more treatments of 125*mc* for 15 minutes with intervals of two days between treatments.

Another period of rest of two months was followed by two more treatments on January 19 and 21 with 125*mc*. From February 3, 1920, to January 21, 1922, she had 85 treatments of 15 minutes duration aggregating 1132 millicurie hours.

In February, 1922, the growth on the cornea looked thinner, and that on the sclera seemed to have largely

disappeared, but there was still an abundant pigmentation of the whole region.

The patient had an abounding faith that she was getting better and would recover. In May, 1922, it was clear that the growth was again increasing and was extending over the cornea from below. The appearance at this stage was much like that depicted in the colored drawing, Fig. 2.

She was then sent to the Physicians' Radium Association of Chicago for treatment under the direction of Dr. Wm. L. Brown, and in the next six months received nine treatments in the following manner:

May 8, 1922. One tube, 100*mgs*, screened with 1.0*mm* of platinum and 3.0*mm* of lead with window in lead corresponding with the size of the lesion was moved slowly about the affected area for fifteen (15) minutes.

May 12, 1922. One full strength plaque 5*mgs*, screened with 0.7*mm* of rubber except for an aperture 5*mm* in diameter, moved over the growth for fifteen (15) minutes at a distance of about 2*mm*.

June 5, 1922. One full strength plaque 5 *mgs*, screened with 0.7*mm* of windowed rubber, with one tube of 100*mgs* on the back of the plaque, the tube screened with 1.0*mm* of platinum, moved over the lesion for fifteen (15) minutes at a distance of about 2*mm*.

June 12, 1922. Treatment of June 5th repeated, 15 minutes.

June 26, 1922. Treatment of June 5th repeated, 15 minutes.

July 17, 1922. One full strength plaque 5*mgs*, screened with 0.2*mm* of aluminum and 0.7*mm* of rubber and a window 5 x 8*mm* to correspond with the size and shape of the lesion, held in one position over the lesion for fifteen (15) minutes, and almost in contact with it.

July 31, 1922. Treatment of July 17th repeated, 15 minutes.

September 8, 1922. Treatment of July 17th repeated, 15 minutes.

October 10, 1922. Treatment of July 17th repeated, 15 minutes. Making nine (9) treatments in all. The full strength 5*mg* plaque which is mentioned is a metal square 10*mm* square, containing 5*mgs* of radium distributed over the surface of this area. The radium plate is held in place by a transparent varnish with so little resistance to the radium rays that for all practical purposes the screening is zero. Where there is mentioned windowed lead in the use of the tube, the lesion was subjected to irradiation through 1.0*mm* of platinum only, except where the tube was used in conjunction with the plaque, in which case, it was

screened with the 1.0*mm* of platinum with the thickness of the plaque in addition. When the full strength plaque was used, the lesion was always subjected to direct unscreened irradiation.

The case was presented at a clinical meeting of the Chicago Ophthalmological Society, May 15, 1922, about the time the last series of treatments was being started. Prof. E. Fuchs of Vienna was present at the meeting, examined the case carefully, and took part in the discussion (*Amer. Jour. of Ophthalmology*, September, 1922, p. 729).

The author expressed the thought that because of the chronicity of the growth, it might not be a melanotic sarcoma, as was supposed, but one of the rare forms of pigmented epithelioma, a view with which Dr. de Schweinitz, who had also seen the case, was inclined to agree.

Prof. Fuchs thought the case a most extraordinary one, but doubted the existence of pigmented epithelioma, and thought such a tumor started from a nevus, which is mesoblastic tissue and hence is a regular sarcoma. He thought the safest procedure would be to remove the eye together with all the conjunctiva containing pigment. He suggested as an alternative that the tumor mass be excised, the surface cauterized with the galvano cautery and treated afterward with radium. If the examination showed the excised tissue was sarcoma, the eyeball should at once be enucleated. If it proved to be carcinoma, it was not so dangerous, although there would be likelihood of local recurrence.

When reminded of the small pigmented spot that had been on the sclera, from which the tumor had grown, he thought that spoke for the existence of a nevus, and that the growth was probably melanotic sarcoma.

The patient again declined enucleation or any operative procedure and the radium treatments were continued.

On July 24, 1922, after six treatments, it was noted that the condition was much improved, and that the pigment, scattered over the sclera and through the conjunctiva, was disappearing.

The last treatment was given October 10, 1922, and by October 31, the growth on the cornea had almost entirely

disappeared. Improvement continued, even the pigment gradually fading away.

At the time the last colored drawing (Fig. 3) was made May 15, 1923, there was no sign of the growth on the cornea, except a faint line seen with a loupe on the inferior temporal quadrant of the limbus and a few peppery spots of pigment here and there in a rather more deeply shaded area of the sclera, which had formerly been almost black. The vision of the eye remains $\frac{20}{30}$, the same as before, and there is no sign of any change in the interior of the globe. The patient's health seems to be perfect, she has gained in the last three years 22 pounds and there are no signs of metastasis.

The prompt response to the last series of nine treatments in which smaller doses of radium metal were used is in marked contrast to the results obtained with larger and more frequently applied irradiations from emanation tubes.

How much effect is to be ascribed to the cumulative effect of such long continued treatments is a question for radiologists, but some maintain that the cumulative effect is not great. It would seem as if the shorter beta rays from the radium distributed over a plaque and brought into closer contact with the surface of the growth were much more effective in disorganizing it than the more deeply penetrating gamma rays. Whether the tumor is destroyed or only dispelled can be answered only by future developments. At present the case has the appearance of being cured. The patient was seen again on May 1, 1924, the appearance is even better than it was in June, 1923. At a distance of two feet it is hardly possible to note any abnormality but on careful study a few small particles of pigment are found scattered in the episcleral tissue.

RETINAL HEMORRHAGES IN BOTHRIOCEPHALUS ANÆMIA.[1]

By Dr. MARTIN COHEN, New York.

THE presentation of a report of this case to the Section is important from two standpoints: not only because of the infrequency of occurrence of cases of bothriocephalus anæmia in this country, but also because the associated retinal hemorrhages are fairly significant, and frequently accompany this affection.

The bothriocephalus is a long, broad tapeworm encountered mainly in Finland, Russia, and Switzerland, from which sections reports are, for the most part, available. In this country they are generally confined to immigrants who give a history of previous infestation.

The eggs, with their embryos develop in fresh water, where they are devoured by certain types of fish. When these fish are eaten in a raw or improperly cooked state, the eggs are apt to become adherent to the human intestinal mucous membrane, where they subsequently develop fully. If the tapeworm, in time, is not completely evacuated, it remains in the intestinal canal and is apt to die there, and segments decompose.

The products of this decomposition, according to some authorities, are absorbed by the intestinal mucous membrane, causing a toxæmia which is fatal to the blood elements, and resulting in a severe anæmia. This anæmia is similar to the so-called primary pernicious anæmia, from a hematological standpoint. In all probability, the retinal hemorrhages are

[1] Read before the Section on Ophthalmology, New York Academy of Medicine, December, 1923.

indirectly due to the same hemolytic agent, which produces the toxæmia.

The first articles published on bothriocephalus anæmia associated with retinal hemorrhages were reported, respectively, by Doctors Gustav Reyer of Dorpat (1) in 1886, A. Natanson of Moscow (2) in 1894, and Tschemolossow of St. Petersburg (3) in the same year, while to the recent literature Doctor Russel L. Hayden of Kansas City (4) has contributed an article describing thirty cases of bothriocephalus anæmia occurring in this country. It is claimed that ten per cent of people infected with this parasite develop severe anæmia and that more than fifty per cent of these cases, according to Natanson (5) show retinal hemorrhages.

The case reported in this communication occurred at the Harlem Hospital on the medical service of Doctors Neff and Bullowa, to whose courtesy and assistance the writer is indebted. Thanks are also due to Doctor A. M. Sala who reported on the laboratory findings.

Case History.—Patient, E. D., female, came to the hospital on Nov. 2, 1923, and was admitted to the medical service. She is seventeen years of age, born in Finland, and has lived in this country one year. Her occupation is that of waitress: she came to the hospital complaining of dyspnœa, general weakness, œdema of the face and lower extremities, also of a moderate diarrhœa and vomiting. Through an interpreter, she stated that she had the yellow sickness which was prevalent in her country and which produced a diarrhœa from which she has suffered intermittently for eight years. During this period she had had a brownish discoloration of her skin. Her father died at the age of sixty-five years of some stomach ailment, and her mother, three brothers and two sisters are alive and in excellent health. The physical examination of the patient was as follows.

Physical Examination.—There was a brownish-yellow discoloration of the skin: patient was drowsy; eyelids, face and lower extremities were moderately œdematous; there were no petechiæ present in the skin; there was one small isolated hemorrhage on the right ocular conjunctiva; a proved functional systolic murmur was heard at the base of the heart: no

glandular enlargements were observed; the spleen was slightly palpable; otherwise her physical examination was normal.

Blood pressure on admission $\frac{110}{50}$; pulse 110; temperature 101°F.

LABORATORY EXAMINATION ON ADMISSION.

Red Blood Cells	700,000
Hemoglobin	15 per cent
Color Index	1 plus
Bleeding Time	4 minutes
Clotting Time	5 minutes
Blood Platelets	30,000
White Blood Cells	5,000
Polynuclears	34 per cent
Small Lymphocytes	53 per cent
Large Lymphocytes	5 per cent
Transitionals	6 per cent
Eosinophils	2 per cent

These findings show a uniform decrease in blood constituents. Megaloblasts, normoblasts and macrocytes were present. Marked poikilocytosis, also granular degeneration.

The Van der Bergh and Fouchet tests both show a hyperbilirubinuria. Blood chemistry of the non-nitrogenous proteins was normal. The serum albumin and globulin was not tested. Urine was normal except for a slight trace of albumin. Wassermann reaction was negative. The ova and bothriocephalus latus were recovered from the stools.

Two transfusions, in conjunction with vermifuge treatment, brought about improvement in the general as well as in the eye condition. The patient is still under treatment at the hospital.

Clinical Diagnosis.—Bothriocephalus Anæmia.

Ocular Findings.—The eye examinations were conducted every three or four days for several weeks, and the findings were practically similar in both eyes. The vision was blurred in both eyes at first, but became normal, following treatment. The vision, functional tests, media and external examinations were normal. The ocular scleral hemorrhage was rapidly absorbed. The fundus changes in both eyes were practically similar, although the left was slightly more involved.

The changes noted were:

The disks had a grayish hue; their margins were completely obliterated, due to œdema which extended to about a disk diameter and more beyond its entire border; the profuse hemorrhages were significant and were located around the disk; none were seen in the equatorial or peripheral areas: the shape of some of the hemorrhages was, at first, circular, then became hemispherical, producing the typical appearance of a pre-retinal hemorrhage.

These pre-retinal hemorrhages had their upper margin straight, sharply defined and the lower border was circular and fringed at several points: the blood vessels disappeared at either border. The microscopical report of this type of hemorrhage has been made by J. H. Fisher (4). Two of these occurred in each eye in the neighborhood of the macula. These hemorrhages were twice the size of the papilla—there were also striated polygnal and circular hemorrhages. Some of the hemorrhages bordered the vessels, mainly the veins: others surrounded the vessels and were large, and were in a stage of resorption showing upper areas of lighter color in them as seen at various examinations. The center of some of the hemorrhages in the early examinations showed the yellowish dot seen in other severe anæmias, disappearing after a few days. These retinal hemorrhages are similar to the petechiæ which occur in the skin, pleura, and in the endocardium in various blood conditions. The retinal veins were moderately engorged and tortuous: the retinal arteries appeared normal: the aqueous and vitreous media were clear. On December 12, 1923, the fundus findings show complete normal fundus of the right eye. The left eye ground showed a fresh isolated pre-retinal hemorrhage located at a disk diameter below the disk. Whether this hemorrhage is due to a recurrence of the condition—as the head of the worm was not yet identified—or is due to the chronic intoxication, continued observance will reveal. No white spots or pigmentary alterations were seen in the fundi. All of the pathological findings of the fundus are probably the result of the venous stasis and are not of inflammatory origin. On December 23, 1923, the patient was discharged from the hospital as cured: the laboratory reports of the blood and the fundi were normal.

Natanson (2) reports post-mortem findings in one case in

which he found œdema of the disk, retinal hemorrhages in the various layers of the retina, also several pre-retinal hemorrhages: no blood was found in the vitreous, the vascular walls showed normal endothelial lining, media and adventitia.

Tschemolossow (3) in one of his cases, found that the retinal vessels showed endothelial swelling of the intima and retinal hemorrhages.

Causation of Hemorrhages.—As to the causation of the retinal hemorrhages and œdema of the papilla and surrounding retina in anæmia, Leber and others have presented valuable suggestions. Recently, physico-chemical studies of the blood in anæmias and allied conditions have shown some additional facts as to the probable causation of œdema and hemorrhages in general. Tallquist (7) in 1907 was able to produce an anæmia simulating a pernicious anæmia by macerating the decomposed segments of the bothriocephalus latus and then isolating a hemolytic substance, probably of a lipoid nature and injecting it into an animal. It is claimed that when the parasite dies the segments decompose, producing a hemolytic substance which is absorbed by the mucous membrane of the intestinal canal and causes the toxæmia which is deleterious to the blood elements and the bone marrow, thus causing the anæmia. In severe anæmia, the proteins, such as fibrinogen, serum albumin, and serum globulin, are decreased and the total solids are also diminished. The fluids are relatively increased, which condition probably decreases the osmotic pressure of the blood and allows water to be absorbed in the surrounding tissues, which would partly explain the œdema present in the skin and fundus oculi. As the proteins are decreased, the viscosity of the blood and the blood pressure become diminished, thus effecting a retardation of the circulation. This fact, I believe, favors a venous stasis and, in toxæmia, this stasis is liable to produce hemorrhages in the skin and fundus oculi by diapedesis, as the hemorrhages are readily absorbed, rupture of the vessel walls never having been reported.

Conclusions.—The important facts to be drawn from this report are:

1. In severe anæmias which simulate pernicious anæmia, the bothriocephalus latus may be the causative agent. Ac-

cording to accounts in the literature, more than one half of these cases show significant retinal hemorrhages.

2. Retinal hemorrhages are numerous and similar in character to those described by Tschemolossow (3) namely: the fundus shows pre-retinal, circular and striated hemorrages, and they are mainly located around the optic disk.

3. Retinal hemorrhages are not pathognomic of bothriocephalus anæmia, but suggestive, if associated with secondary anæmia of unknown origin.

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POSTERIOR SCLEROTOMY WITH PERMANENT DRAIN FOR RETINAL DETACHMENT.

By Dr. MEYER WIENER, St. Louis.

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ACCORDING to de Wecker (1), the first sclerotomy was performed by Guerin of Lyons, who employed puncture of the sclera for the relief of tension in glaucoma as early as 1769. This antedates by nearly a century, the work of Kittel (2), to whom Uhthoff gives priority. The latter published an article on puncture of the sclera and retina for retinal detachment, in 1860, but several others besides Kittel had already performed sclerotomy for either retinal detachment or glaucoma. James Ware (3), in 1805 advocated scleral puncture for the relief of retinal detachment. Mauthner (4), quoted Middleton and Ritchie as having done posterior sclerotomy in 1853 and said that Hancock (5) in 1860 had first made a sclerotomy with a knife or needle, and then passed the instrument through the ciliary muscle. Von Graefe (6) in 1863 published a report on fifty cases of retinal detachment where he opened the sclera and also punctured the retina, his plan being to permit access of the subretinal fluid to the vitreous cavity. His results were not very satisfactory. He obtained immediate relief in about one half of his cases, but there was permanent improvement in only four cases, eighteen months after operation. Argyll-Robertson (7) is generally credited with being the first to trephine the sclera for increased tension. having reported four cases in 1876, with good results in three of them. He later, however, abandoned the operation as being of little permanent value. However, de Wecker (8)

four years before, in an article published in the *Annals d'oculistique* on "Uses of the Ocular Trephine" says, "In cases of glaucoma absolutum, where there is extensive atrophy of the iris preventing excision of a portion of it, and where simple sclerotomy will not suffice, in order to relieve pain, and to avoid enucleation, I remove a disk 1.5*mm* in diameter next to the corneal border, avoiding the lens, and not too near the ciliary body. I establish by this means, a large filtrating cicatrix." This undoubtedly gives precedence to de Wecker as being the first to trephine the sclera for glaucoma. De Wecker himself refers to an article written by Anagostakis (9) in 1870 who credits the ancients as having used the ocular trephine, although he does not state for what purpose. Czermak (10) believed that scleral trephine gave no satisfactory results and soon abandoned it.

While it is true that very few permanent good results have been obtained from scleral puncture, trephining, or in fact, any other means of relief of detached retina, quite a number of authorities have reported occasional good effects from opening the sclera over the detached area. De Schweinitz (11) has obtained good results from scleral puncture when followed by salt injections and rest in bed. McMullen (12) also reported a cure after repeated scleral punctures. Wolfe (13) advised long incision in the sclera made obliquely and favoring their keeping open longer. Galezewski (14) realized that nothing short of a permanent drain would suffice to keep the retina in place. He therefore passed a needle threaded with very fine gold wire through the conjunctiva, scleral wall, choroid, and making a counter puncture, tied the ends over the conjunctiva. Infection following the gold thread, always resulted in loss of the eye. If he had tied the threads under the conjunctiva, or left them loose, covered by conjunctiva, he probably would have effected a cure in many cases. He later attempted to suture the retina to the choroid with catgut, as Martin (15) and Evers attempted with horse hair and silk.

Deutschman (16) claimed for his bisection operation published in 1895, reduction of the tension on the shrunken retina, which, according to his experience may be so marked that re-attachment is impossible, unless several incisions are made in the region of the ora serata. The results obtained by

Deutschman were most brilliant, but others have not been so successful with the same methods.

Mueller (17) believing that many cases of detachment were due to the disproportions of the shrinking vitreous and the ocular outer coats, resected a piece of the scleral wall. He first performed a Kroenlein operation and dividing the external rectus, excised a biconvex strip of sclera 20*mm* long and 8 to 10*mm* wide, with its length parallel to the equator.

Parker (18) trephined three cases of retinal detachment in order to provoke displacement. In the first two cases he did not incise through the choroid and retina, and the results were not so good. In the third one he used a cataract knife to divide the retina and choroid. A marked reaction followed, but subsided, and after ten days the retina was re-attached; eight months after operation the vision had increased $\frac{4}{60}$ to $\frac{6}{12}$ with a nearly normal visual field. Tiffany reported a similar successful case. He removed, however, a piece of choroid with the scissors. Later Parker (19) reported fifteen operations on eleven patients. Vision was improved in four and unimproved in five and in three made worse. In one case vision was increased from $\frac{4}{60}$ to $\frac{6}{15}$. An additional case was reported in 1917.

Thompson and Curtin (20) advocate scleral trephine and after an interval of about ten days to aspirate the subretinal fluid and repeat, if necessary, so long as the scleral opening remains patent. They reported three cases, showing improvement in two, and none in the remaining one.

A case of very rapid improvement with increase of vision from $\frac{6}{60}$ to $\frac{6}{6}$ was reported by Walker (21) as the result of five scleral punctures with a discission needle 10*mm* posterior to the lower border of the cornea. The retina was pierced at each puncture and the last was turned ninety degrees to let out the subretinal fluid under the conjunctiva.

I have been of the opinion for some time that in retinal detachment, where the retina is still free from rents or tears, an operation which would permanently drain the subretinal space without offering any undue tendency toward infection, would offer the best possibilities for re-attachment, in the light of our present limited knowledge regarding the cause and relief of retinal detachment. Posterior sclerotomy by means of the

trephine, as first suggested by de Wecker, and later more successfully carried out by Parker, Curtin, Thompson, Walker and others, is the safest of the methods thus far employed. The chief objection, early recognized by Galezewski, seems to be the almost certain eventual closure of the scleral opening, as observed by Ewing in his report of repeated scleral trephining in a case of glaucoma.

In Ewing's case the tension was 57*mm* with vision of $\frac{6}{180}$ in the beginning. This rose to $\frac{3}{60}$ after use of eserin with almost normal tension, but soon relapsed. After repeated trephinings, the eye became perfectly blind with final tension of 70*mm*, although there was no pain. He showed that the scleral opening always eventually closed. Hardy, in the discussion on this paper, stated that he had performed a number of operations through the sclera by various methods and had found that it was impossible to keep the scleral wound open, that they remained open a week, but filled in with dense fibrous tissue and at the end of a month, all were closed.

A method was sought which would keep the scleral window patent without causing irritation, and offering the least possible danger from infection. Horse hair, to the writer, seemed to answer this requirement. Surgeons doing plastic work about the face have long recognized that horse hair, in making fine cutaneus approximations, produces fewer stitch abcesses than other suture material. The fact that it is an animal product, which can be thoroughly sterilized, suggests its tolerance by the tissue. Its stiffness permits it to be easily handled in slipping it into the scleral opening. The plan contemplated, after removing the scleral button and puncturing the choroid, was to introduce a piece of horse hair about 8 to 10*mm* long, obliquely through the trephine opening into the space between the choroid and retina. One half is placed in this space. The other half which protrudes upon the outer surface of the sclera, is held in position by the replaced conjunctival flap. This plan was carried out in the first few cases operated upon.

On account of the difficulty of technic in placing the piece of hair in proper position and keeping it there, the technic was later modified. Two trephine openings are made in the sclera with a 1.5*mm* trephine, 1.0*mm* apart. A sharp, full curved

needle, previously threaded with a strand of medium sized horse hair, is passed into one opening, and out of the other, endeavoring to pass through the space between the choroid and retina. The suture is then clipped off, permitting about 1.5*mm* to overlap the sclera beyond each trephine opening. The conjunctival flap is then replaced with two or three silk sutures.

CASE 1.—C. F. S. Age 53; first consulted me March 28, 1914, for a detachment of the retina of the left eye, which had suddenly come upon him while on a visit to New Orleans in December, 1912, where he had been treated by Dr. Bruns, and later Dr. Feingold. There were then a few floating spots in the right vitreous, but no sign of detachment. O.D.V. − 8.0 D.S. ⊃ − 0.5D. Cyl. ax O = $\frac{20}{20}$. By October 23, 1915, his myopia had increased 0.75 diopter. By October 25, 1916, it had increased 0.5 diopter and the spots in the vitreous seemed more numerous. I advised abstinence from salty foods, and prescribed dionin locally and K.I. internally. Condition remained about the same until February 9, 1917. While on a visit to New Orleans he stubbed his toe violently against the walk, and that evening noticed a blurring of his sight. The next day, Dr. Feingold examined him and found that he had a beginning detachment. He came home immediately and went directly to the Barnes Hospital, where he was examined, with the following findings:—

Left eye, complete detachment with secondary cataract and posterior synechiæ. No perception of light. Right pupil dilated with atropine. Detachment of retina involving lower nasal quadrant and extending to the equator and just below the disk. Grayish reflex with few folds. Central vision almost unimpaired, but upper and outer fields contracted to about twenty degrees. Patient was placed in bed with a tight bandage. December 16th, detached area a little more gray and slightly more extensive. Two per cent. subconjunctival injection of sodium citrate was given and tight bandage reapplied. December 17th, 18th, and 19th showed no change. On December 20th and 21st he was worse and a trephine operation was accordingly advised.

December 22, 1917, under cocaine anæsthesia a conjunctival flap with apex 4*mm* from the cornea was made in the lower nasal quadrant, keeping close to the sclera. In the middle of this triangle, a 2*mm* trephine opening was made in the sclera, the button being removed with scissors. Incision in the choroid was made with a Graefe knife and a large

amount of subretinal fluid evacuated. The flap was replaced with three silk sutures and a tight bandage applied. Just before applying the bandage the patient thought he could see better in the upper outer field. December 26, 1917, dressing was removed and the retina found almost completely attached. It remained so with normal field and no distortion until January 3rd, when the patient observed a slight distortion in the ceiling wainscoating, although nothing could be seen with the ophthalmoscope. January 6th, a slight gray reflex could be made out in the extreme lower periphery, which gradually increased until January 20th, when the lower half of the retina was found detached, but not quite so much as before the operation.

On January 21st, another trephine opening was made in the lower outer quadrant, a small puncture made in the choroid obliquely with the Graefe knife, a large amount of subretinal fluid escaping. The flap was replaced with three silk sutures, and a tight bandage applied. On January 25th, the dressing was removed and the detachment was found considerably worse than before the second operation. Two pearly white bands were seen in the retina in the lower inner quadrant. The higher point of the detachment seen best with 7.0 D. No change on 27th or 29th. On 31st, he seemed slightly better. On February 10th he was quite a bit better, with field of vision unusually good, even in detached area. The eye was sore, probably from pressure which had been kept up, and the bandage was therefore left off and the patient permitted to get up.

On February 12th, as he was no worse, he was permitted to leave the hospital. He continued to improve, although he was without treatment, and was able to go alone to his place of business. The field of vision increased and central vision improved to $\frac{2}{4}\frac{0}{0}$. On March 15, 1918, P.M., his eyes seemed so good, he disobeyed orders about abstaining from sudden or violent movements and swung his Indian clubs freely. He awoke next morning and noticed that the curtain string was not hanging straight. By noon he could barely distinguish light. That afternoon I punctured the trephine opening in the lower nasal quadrant with a Ziegler needle, evacuating a large amount of subretinal fluid under the conjunctiva. The entire retina was detached excepting a small area in the upper nasal quadrant, the upper outer quadrant showing the retina hanging in large folds, which, however, seemed better after the puncture.

A trephine operation was performed again on March 18th, again on March 28th, with no relief. May 17, 1919, three trephine openings were punctured with a Ziegler needle with no improvement.

On May 17, 1919, the conjunctival flap was dissected down, exposing the trephine openings. A piece of horse hair was introduced into one opening and out of the other and clipped off, replacing the conjunctival flap. No improvement was noted. The patient, at the present time (1923) has no perception of light.

CASE 2.—C. D. Age 45. Admitted to Barnes Hospital for operation for inguinial hernia on October 2, 1919. Found to be blind.

Examination showed cataract, both eyes. Right eye; small irregular pupil, calcareous lens, adherent to iris and dislocated slightly downward. Left; lens cataractous, dislocated slightly downward. Marked iridodonesis both. No perception of light on the right. Left: good perception and projection of light. Family history negative.

Patient's history. General health good. Measles, smallpox, at 4 years had pneumonia followed by "brain fever" which produced partial blindness both eyes. No typhoid or malaria. Pleurisy "long time" ago. No trouble since. Occasional fainting spells. No tobacco or alcohol. Weight 120 lbs., constant.

Present trouble. Previous to "brain fever" sight was good. Vision became very poor, right worse; but was able to distinguish objects very close. Tried various oculists, but with no help. In 1900 blow in right eye by piece of wood was followed by cataract and blindness. In 1909 cataract noticed on left, and finally, after gradual failing vision was unable to distinguish objects with either eye.

Was operated upon for his hernia on October 4, 1919, and as he was progressing nicely was taken to operating room for removal of cataract from left eye on October 10th.

Cocaine anæsthesia. Fisher retractors used. Large corneal incision with Graefe knife, fluid vitreous escaping through opening before incision was completed; deep iridectomy made, spatula introduced behind lens, pressure made on cornea below with strabismus hook, and lens expressed in capsule without further loss of vitreous. The method used was that advocated by Col. Smith for removal of dislocated lens. Dry dressing applied and double Fuchs mask. Patient says he does not remember ever having seen so well. Healing without incident. November 1, 1919, iris clear, pupil black and slightly drawn up. O.S.V. + 5.0 D. S. = $\frac{20}{20}$, fundus shows a few small floating spots in vitreous. Disk normal in color and outline, vessels normal. No choroidal spots could be seen. Patient was discharged November 3, 1919.

Patient readmitted to hospital November 28, 1919, with

following history:—Two weeks before on November 14, 1919, saw black spots in motion over his eye. This was intermittent until November 27, 1919, when "a scum" obscuring sight over nasal half of field appeared. This has not been of constant density being more dense now than four hours ago. Examination showed detachment of upper two thirds of the retina hanging down like a heavy water bag. V = $\frac{3}{200}$ in upper visual field. Physical examination revealed chronic tonsillitis, but no pus. X-ray showed three abscessed teeth, which were removed. The eye, however, continued to get worse. Operation was advised. December 22, 1919, under cocaine anæsthesia, traction thread was introduced in conjunctiva at upper corneal margin and eye pulled downward.

A triangular conjunctival flap was made with the apex at the upper outer corneal margin directed backward, and a *2mm* trephine opening was made in the sclera about *10mm* from the corneal margin. The choroid was grasped with iris forceps and punctured with the point of a Graefe knife, allowing free escape of subretinal fluid. A piece of sterile horse hair *1cm* long was placed in the opening so as to rest half in the space between the retina and choroid, with the other half overlapping the scleral opening, being held in place by the replaced conjunctival flap.

December 5th, dressing removed. Illumination with ophthalmoscope shows vitreous opacities, but a red reflex from every part of the fundus field. Retina seems to be entirely in place and patient able to count fingers even in lower field, which had been perfectly blind before the operation. December 6th, V = Fingers at 4 feet. December 10th, V = Fingers at 5 feet. The condition remained the same until December 29th when the patient developed influenza from which he recovered and was discharged February 6, 1920.

Case 3.—C. J. Age 55. Had been under my observation for many years for high degree of compound myopic astigmatism. Vision had never been better than $\frac{1}{2}\frac{8}{0}$ in each eye. On December 28, 1920, he came complaining of seeing a spot before the right eye, observed for several months. Sight had been badly blurred for more than a week. ODV = $\frac{3}{200}$ in lower part of field. OSV − 6.5 D.S. ⊂ − 3.0 D.Cyl. ax. 170 = $\frac{1}{2}\frac{8}{0}$. There was an extensive detachment of the right retina, especially below and nasally. Field of vision limited to a small section in the lower nasal quadrant. December 29, 1920, a scleral trephine was performed above and temporally in the right eye, well back of the limbus. A piece of horse hair was slid in the opening between the

retina and choroid, after first opening the choroid with a Graefe knife, letting out the subretinal fluid. Conjunctival flap was then replaced with two silk sutures. The detachment decreased slowly until January 18th, when the field was twice the size it had been before operation and vision $\frac{10}{300}$. From this time, however, it began to grow worse and the conjunctiva around the horse hair became red. On February 2, 1921, ODV = $\frac{3}{180}$. On February 26th, the hair was sticking through the conjunctiva and was easily pulled out, the scleral wound having closed. On March 9, 1921, another trephine operation was done, this time making two openings and threading the hair into one and out of the other. The vision, however, steadily decreased while the detachment increased with gradual clouding of the lens. When last seen on December 28, 1922, he had a well developed cataract with perception of light only, below and to the temporal side. OSV = $\frac{1}{2}\frac{0}{0}$ with correction.

CASE 4.—F. E. Age 61. First seen December 9, 1920, complained of gradual failing vision, left eye, but suddenly much worse three days before. ODV = 1.9 D.S. $\frown$ + 1.5 D.Cyl.ax 0 = $\frac{1}{3}\frac{0}{0}$. OSV = fingers at nine inches in upper field. Field of vision limited to small sector in upper temporal field. Right fundus showed slight cloudiness of lens. Left showed extensive retinal detachment in upper temporal portion. Patient refuses operation. Used Sodium Citrate injections without improvement. June 10th was sent to the Jewish Hospital for operation. A double trephine was done in lower inferior quadrant. There was never any reduction in the detachment, but a steady progress for the worse. He was last seen November 20, 1922. His lens was cloudy, so as to obscure a view of the fundus. There remained bare perception of light in the upper temporal portion of the retina.

CASE 5.—Wm. I. J. Referred by Dr. Mann of Texarkana, Ark. Age 40. First seen June 28, 1922. Lost sight of right eye by injury when 14 years of age. No perception of light and eye converges. Left eye injured by glass from auto wind shield in 1919. Penetrating wound of sclera with hemorrhages in vitreous which healed with $\frac{3}{2}\frac{0}{0}$ vision resulting. Had noticed floating spot in eye since injury, but 4 months ago, vision in looking downward was diminished. Right pupil eccentric, iris atrophic. Left shows scar in sclera in extreme nasal quadrant, just above horizontal plane about 2.5*mm* long, and jogged in character. OSV = $\frac{1}{1}\frac{5}{0}$. Fundus showed extensive detachment of retina below, extending nasally just above the horizon

and temporally also. Retina in deep folds similar to high waves on the ocean. Only the central and part of the nasal field remaining. June 29th was operated upon at the Jewish Hospital. A conjunctival flap was dissected back in the nasal quadrant. Two 1.0*mm* trephine openings were drilled in the sclera well back toward the equator. A needle threaded with horse hair was passed into one through the subretinal space, out of the other and the ends clipped off, 1*mm* beyond each trephine opening. A thick fluid exuded with the exit of the needle, conjunctival flap replaced with two stitches and dry gauze dressing applied. Retina immediately fell back partially and on July 9th was reattached, excepting the lower nasal portion over the sight of the old scar. Two days later the retina began to separate. On July 13, 1922, under twilight sleep an attempt was made to cut across the vitreous to separate any band emanating from the old scar. No hemorrhages in the vitreous resulted when examined two days later, and the retina was in slightly better position. On July 21st, he was worse, the vision having been reduced to $\frac{20}{100}$ when he was discharged from the hospital and permitted to go back to Arkansas.

On September 20th, I received a letter from Mr. J., typed by himself, stating that he was much better, that he was able to read half way down the chart and that his field seemed larger. On October 13th, the patient wrote that three days previously he had suddenly lost his central vision completely. My latest information was to the effect that he had remained blind.

Case 6.—T. L. Age 37. Referred by Dr. F. P. Parker. First seen in consultation April 27, 1922, with large detachment of outer half of retina of right eye. Field of vision as shown, nasal half missing. Visual acuity right $= \frac{4}{300}$. Left $= \frac{1}{2}\frac{6}{0}$; with $-$ 4.0 D.S. Highest point of detachment seen with + 9.0 D.S. On May 22, 1922, conjunctival flap dissected back from inferior temporal quadrant with apex, next to the cornea. Two openings drilled in sclera about 10*mm* back and 1*mm* apart. Needle threaded with horse hair, passed into one opening and out of the other, through the subretinal space, fluid exuding. Hair clipped off overlapping 2*mm* on each side, conjunctival flap replaced with two silk sutures. On first dressing patient could see better, with absence of wrinkled appearance of bag. Retina seemed to be reattached except at a point just to the temporal side of the macula. Highest point of separation measured + 6.0 D. Small hemorrhages at sight of trephine. On May 29th, patient left the hospital. May 31st, ODV $= \frac{7}{200}$. Peripheral field for white about normal. June 1,

1922, ODV − 4.0 D.S. = $\frac{1}{100}$. June 8, 1922, ODV = $\frac{1}{80}$. Mass in fundus seems thinner. June 12, 1922, − 4.0 D.S. = + 0.75 D. Cyl. ax. 135 = $\frac{1}{80}$. Exudate and hemorrhaging absorbing. June 29, 1922, − 4.0 D. S. ⊃ − 1.0 D. Cyl. ax. 90 = $\frac{1}{80}$ and few of $\frac{1}{40}$. This is the best vision patient has attained in this eye. The last examination made January 18, 1923, vision was only $\frac{1}{70}$, but retina was entirely reattached with normal field.

CASE 7.—T. L. M. Age 59. Referred by Dr. Haeberle. First consulted me on November 6, 1922, on account of seeing streamers before right eye. ODV = $\frac{1}{40}$ + 1.25 D.S. ⊃ − 0.5 D. Cyl. ax. 90 = $\frac{1}{20}$. OSV = $\frac{1}{80}$ + 1.25 D. S.⊃ − 0.5 D. Cyl. ax. 90 = $\frac{1}{20}$. Fundus reveals a few floating spots in right vitreous with an extensive exudate in upper temporal quadrant near periphery. On January 7th, dilated pupil with dextrohyoscyamine. The area of exudate was seen to be much more extensive with retina in folds in places. Advised X-ray of teeth. November 8, 1922, X-ray shows pus pockets at the roots of two teeth, which were immediately extracted. The following day his eye seemed better. On November 13th, came with a history of having observed a sudden blur over the right eye the day before and being hardly able to see. Examination showed detachment of retina of upper and temporal quadrants of right eye. Vision reduced to $\frac{1}{300}$. Corresponding absence of lower nasal field. Patient seemingly in good physical condition. Sent to Deaconess Hospital, where he was placed in bed, given pilocarpin sweats and laxatives. Also tight bandages applied. Physical examination showed B.P. 110−60. Many hyalin casts, but no albumin. Otherwise negative physical examination. Right fundus showed numerous floating spots in fluid vitreous. Retina separated above and temporally, hanging down like a large bag. Disk still seen with indirect method, but obscured by overhanging retina with direct. Injections of Sodium citrate were given every third day. As there was no improvement after two weeks of treatment, operation was performed November 28, 1922. Local anæsthesia. Speculum introduced; eye fixed, conjunctival flap with apex near upper temporal corneal margin dissected up and out, to near the fornix, where two small openings were drilled through the sclers, 1*mm* apart. As the needle, threaded with horse hair, was passed in one opening and out of the other, considerable subretinal fluid escaped. Ends clipped off, overlapping scleral 1.0*mm*. Flap replaced with two silk sutures. Examination with the ophthalmoscope showed the retina to be almost altogether in place. Dry dressing applied. The retina re-

mained in place and the field of vision intact until December 6th, when a single fold of retina was seen to be separated in the upper temporal periphery. This remained stationary until December 12th, when it was found to be much more extensive with a large preretinal hemorrhage below the disk. On December 12th, the detachment was hanging down like a bag as before the operation. The process continued on December 11th, the field of vision was limited to the upper half of temporal area. On December 12th, patient was taken to operating room, the conjunctival flap was dissected as before; the horse hair was removed and reintroduced, evacuating considerable subretinal fluid. Ophthalmoscopic examination was not encouraging, only a very slight improvement being noticed. No material change was noticed in the patient's condition and he was discharged from the hospital December 20th. The area of detachment seemed to gradually get thinner. On February 12, 1923, the retina still hung down like a bag, but the area below was flatter. February 15, 1923, ODV = $\frac{8}{200}$. Field large. February 19, 1923, area of detachment almost all resolved, except for a small fold near the macula. ODV = $\frac{10}{200}$. The last examination on March 13, 1923, ODV = $\frac{10}{200}$. Field of vision almost normal peripherally, but scotoma corresponding with detached area.

A summary of the seven cases reported shows complete reattachment in two cases with greatly improved vision following the operation. Case seven is still under observation, and while he is worse now than at the time of operation, he is better than he was a few weeks ago and still has some chance of improvement. The other four cases either received no permanent benefit or were made worse following the operation. Only one case has shown intolerance of the drain. This was probably due to it being placed poorly, but since the second operation it has remained in place without irritation. The method has been used in several cases of glaucoma, which will be reported later. In each case reported above, a thorough physical examination, including search for focal infection, has been made, and every possible effort made to remove cause before operation.

CONCLUSIONS.

The advantages of this operation for retinal detachment embrace all of those possessed by the simple trephine opera-

tion, plus the superiority of permanent drainage. It is simple and easy of execution, with very little or no pain, when thoroughly anæsthetized. Failure from one operation does not preclude the possibility of a second or third operative attempt. Its inadequacy is felt in cases where there are vitreous bands or where the space between the choroid and retina is filled with a thick organized substance. In the former it could be combined with Deutschman's bisection operation or some similar method.

A serious shortcoming is the resulting reduction in the usually already lowered tension. This is especially grave where the case is complicated by cataract, as less assurance of ultimate favorable visual results must be prognosticated.

The method is offered as a suggestion, which seems to offer enough promise to justify further trial.

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REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. BEN WITT KEY, Secretary.

MEETING OF DECEMBER 17, 1923. DR. ELLICE M. ALGER, CHAIRMAN.

Dr. J. S. Somberg presented a case for diagnosis of possible **foreign body** in the **globe**. A man, aged 35 years, was struck in the left eye by a chip from a metal clamp. Three days later he noticed some dimness of vision and fly-like specks before the affected eye.

Examination: The right eye was normal. The left eye showed no evidence of injury; the cornea and iris were normal; the pupil was slightly irregular but the reaction was normal. Vision = $\frac{20}{70}$, with correction = $\frac{20}{20}$. Vitreous opacities were present. At the nasal side of the disk there were several patches of chorioretinal change, and in this area there projects forward eight diopters into the vitreous a regular column or mass, very distinctly seen standing forth in relief. The sides of this rounded column are greenish in color and irregular. The anterior portion of it is slightly rounded, with two small areas of pigmentation upon it. The column measures about one disk in diameter. The field of vision shows a scotoma corresponding to the choroiditis.

This peculiar mass in the eye increased in height about two diopters in three months. X-ray examination was negative.

Dr. Somberg regards the case as one of retained foreign body in the globe which does not cast a shadow and cannot be attracted by a magnet.

Dr. Martin Cohen reported on a case of **Retinal Hemorrhages** in **Bothriocephalus Anæmia.** This case report is published in full in this issue.

Dr. BEN WITT KEY presented a case of **Embryonic Zonular Opacity of the Cornea.** A man, aged 30 years, received a minor injury of the cornea of the right eye two weeks before coming under observation. Family and previous medical history were negative. In the superficial layers of the substantia propria of the cornea there was a disciform arrangement of what appeared to be fine yellowish-brown granules, equally and regularly distributed over the entire pupillary area and covering a portion of the iris area for an extent of about 1*mm*. This area was sharply delimited by a faintly white crayon-like line of opacity, drawn about the margins of the granular zone. Under the slit-lamp corneal microscope these granules appear as round, slightly yellowish (almost white) refracting bodies. They possess no light reflecting surfaces. Some are smaller than others, ranging from one to three times the size of a red blood cell, each standing out alone in clear corneal substance and remarkably distributed equidistant from each other. The posterior layers of the corneal stroma were clear, and the posterior corneal surface was free of any deposits. The anterior chamber, aqueous, iris and pupil were normal. The vitreous and fundus were also normal. Examination of the left eye was negative in every detail. Vision of the right eye = $\frac{7}{10}$, corrected to $\frac{10}{10}$ by -1.25 c 90; vision of the left eye = $\frac{10}{10}$, corrected to $\frac{10}{10}$ by -1.25 c 75.

The physical examination was negative, and the laboratory reports negative except that the cholesterol content of the blood = 240*mg* per 100*cc*. All treatment over a period of ten months produced not the slightest appreciable change in the corneal defect.

The lesion could not be classified, but was differentiated from disciform keratitis, corneal degenerations (hyaline deposits, family degeneration, nodular keratitis), hematoidin deposits from hemorrhagic infiltration; corneal lesions associated with systemic disease, such as Addison's disease, dermatitis (icthyosis), multiple sclerosis, cirrhosis of the liver, pancreatic disease; local staining from deposits of chemicals described as the result of treatment or accident; and finally congenital abnormalities.

The author concludes that this is a case of zonular opacity of the cornea made up of slightly pigmented cellular bodies,

embryonic in origin, and analogous to congenital zonular opacity of the lens.

Dr. BEN WITT KEY presented a case of **Embolism** of the **Central Retinal Artery,** in which **Paracentesis** was performed.

A woman, aged 45 yrs., awaked from a night's sleep, totally blind in the left eye. Her previous medical history was of no importance. Examination revealed the characteristic fundus picture of embolism of the central retinal artery in the affected eye. The entire retina was ischemic, the arterial trunks collapsed and fine drops of blood at intervals in the vessels could be observed to push forward a little with each beat of the heart. One small hemorrhage below the disk was noted. The fovea was "cherry red."

Since it was evident that the embolus had occurred only a few hours previously, a paracentesis was performed at once by means of a keratome incision with a large conjunctival flap. The eye-ball was then massaged for a brief period. The ophthalmoscopic examination now revealed no change except that two other small retinal hemorrhages appeared in the lower fundus. Atropine was instilled and the eye bandaged.

Subsequent observation of the case disclosed a rapid clearing of the retinal œdema. On the following day partial filling of the arteries was noted; and in two more days the ischemia of the retina had almost entirely disappeared. The macula appeared normal in five days—and vision = $\frac{20}{200}$. There was concentric contraction of the field of vision of fifteen degrees in the temporal half; twenty-five degrees contraction in the nasal half; and a central scotoma of ten degrees about the fixation point. The nerve-head is a trifle pale with corresponding diminished capillarity, and has remained so to this date.

The case seems to illustrate that when these cases can be seen early there is the possibility that prompt measures may partially relieve and in rare instances entirely relieve an otherwise hopeless condition.

Dr. ARNOLD KNAPP presented a patient who had a **Gumma of the Choroid.** E. C., female, colored, 26 years of age, presented herself at the Herman Knapp Memorial Eye Hospital on October 29, 1923, on account of pain and loss of sight in the left eye, which had been present for ten days. V = $\frac{20}{200}$. There was an area of episcleral congestion up and in which

was tender. The media seemed clear and the pupil dilated fully to atropine. In the upper and inner quadrant of the eye ground there was a rounded solid detachment of the retina measuring 9 D. in elevation. The underlying mass was grayish-brown in color, not mottled and without its own blood vessels. The vessels in the overlying retina showed minute varicosities. Wassermann 4+. The patient was given mercury by inunctions, calomel by mouth and later salvarsan. One week later inflammatory symptoms appeared in the media. The slit-lamp revealed that the lower third of the posterior cornea was covered with thin film-like fibrin and few deposits. The aqueous contained pigment and grayish cells. The anterior surface of the lens was covered with pigment deposits. The vitreous was studded with pigment and coarse granular, nodular formations. November 14th: The aqueous was more hazy, the retina was diffusely cloudy, the retinal vessels congested. The solid detachment was smaller but was not so readily seen. Swelling +6D. Shallow detachment beyond the tumor. November 19th: Swelling was +5D. There were some rounded deeply placed hemorrhages in the retina below the tumor. November 30th: Vision improved to $\frac{4}{10}$. The tumor had nearly disappeared, the retina was œdematous, there were hemorrhages and whitish streaks in the retina. December 15th: V=$\frac{4}{10}$. The site of the tumor showed a flat brownish-gray area of choroidal atrophy of 5 × 6 disk diameters surrounded by a margin of retinal sclerosis with numerous punctate pigment dots in the overlying retina. The retinal circulation was normal except one arterial branch which was very tortuous.

In a symposium on intra-cranial disease, Dr. WARD A. HOLDEN presented a paper entitled, **Papilledema, its symptoms, diagnosis** and **course,** which is published in this issue of the ARCHIVES.

Dr. CHARLES A. ELSBERG spoke on the **Surgical Treatment of Intracranial Disease** with special reference to its **influence** on **papilledema** and **visual disturbances.** This paper is published in this issue.

DISCUSSION: Dr. W. P. EAGLETON stated that unfortunately in his experience he had not found papilledema in either eye of any importance in localizing the side on which the tumor

occurs. He referred to a case observed a few years ago in which the nerve heads were normal, but in the course of a week there was a choked disk of four diopters, and there was a tempero-sphenoidal lobe tumor. These are common tumors and are not apt to show fundus changes, but rather hemianopsia. This is an indication to the ophthalmologist of the importance of taking fields of vision in any suspicious condition of this sort.

Dr. Eagleton sounded a note of warning as to lumbar puncture. He has seen four deaths in one year from lumbar puncture. It is dangerous in tumors of the tentorium because of the stretching on the blood-vessels by sudden displacement causing hemorrhage in the pons, and the patient dies in the course of a few hours. He therefore suggests that the least amount of fluid should be removed for diagnostic purposes.

Dr. E. M. ALGER asked for advice as to what the ophthalmologist should do in order to make an early diagnosis of these cases.

Dr. C. A. ELSBERG stated that it would be important to study a large number of cases carefully comparing the degree of papilledema in the two eyes with the physical conditions found at operation or at autopsy.

In his experience he has not seen any accident from lumbar puncture in posterior fossa lesions, although he was cognizant of the fact that many such cases were reported in the literature. While he thinks that the danger of the procedure is greatly overestimated if the amount of fluid was small (5–10*cc*) and was allowed to run out very slowly, notwithstanding this lumbar puncture in posterior fossa lesions is not without its dangers.

BY DR. DAVID H. WEBSTER, SECRETARY.

MEETING OF JANUARY 21, 1924. DR. BEN WITT KEY, CHAIRMAN.

Dr. LEWIS W. CRIGLER presented a case of **Unilateral Choked Disk relieved by Sinus Operation.** A girl aged 9 came under his observation one week after she accidentally discovered the vision of her left eye was defective. Examination showed O.D. vision $\frac{20}{20}$, O.S. vision p. l. pupil sluggish. Fundus: disk swollen 4.D., some hemorrhages, vessels tortu-

ous. History negative, laboratory tests and X-ray negative. Rhinological examination negative. In view of the rapidity of development the ethmoids were exenterated by Dr. Robert E. Buckley, and although only slight congestion could be demonstrated, a culture showed streptococci, staphylococci and micrococcus catarrhalis. The swelling of the nerve began to subside immediately, and in three days the outlines of the disk could be made out. To-day, at the end of five weeks following operation, the disk showed post-neuritic atrophy and the vision had returned to fingers at six feet.

DISCUSSION: Dr. ROBERT E. BUCKLEY stated that in this case he was able to open one cell at a time, and that he found no demonstrable involvement, that this was one of many cases in which the eye condition improved rapidly, although X-ray findings were negative, and no visible lesion could be seen at the time of operation.

Dr. RAYMOND C. DODD presented the history of a case of **Double Choked Disk, with Recovery following Trauma.** Girl aged 7, in November, 1921, complained of paroxysmal headaches, vomiting attacks, unsteadiness in walking with frequent falls. Past history negative, except for a fall when three years old. Present illness dates from 1918 when four years old she began to have vomiting spells in the morning before dressing, which became more frequent and severe as time went on. In 1919 first noticed unsteadiness of gait, although under the care of general physicians the symptoms became more distressing, and in 1921 the patient was brought to the office of Dr. Broughton and Dr. Dodd. Examination showed V=$\frac{20}{20}$ each eye, pupils normal, nerve heads normal, retinal vessels tortuous. On May 8, 1922, the symptoms increased. V=$\frac{20}{20}$ each. Fundus showed blurring of disks, the vessels were full and tortuous. On May 10, Dr. F. Tilney made a tentative diagnosis of right cerebellar tumor. An X-ray of the head was made by Dr. A. H. Busby, who reported "the changes noted in the internal table of the cranial bones appear too general to be caused by local tumefaction." In June an exploratory laparotomy with removal of appendix was performed, and while in the hospital the child had no attacks. In July a paresis of left internal rectus developed. On July 31, Drs. Tilney and Elsberg advised operation, as soon as the

child recovered from the laparotomy. On August 17, Vision was $\frac{1}{10}$ in each eye, the disks were swollen 3 to 4D. Small hemorrhages at summit of disks. That evening while asleep the girl fell from a settee to the floor, a drop of two feet, striking her head. She suffered intense headache followed by deep sleep, and for two days was listless and feverish. She awoke on the third day feeling perfectly well and has remained so since. The diplopia has disappeared and the disks gradually returned to normal. On October 25, 1923, the eyes were normal to every test. Dr. Dodd concluded that this was a benign cyst, either sub- or epi-dural, the wall of which had become so thin that it was ruptured by trauma.

DISCUSSION: Dr. Homer Smith thinks that it was a tuberculous condition which underwent spontaneous cure. He had had a similar case in which the child died. Dr. Somberg asked if lumbar puncture had been done? Dr. Dodd replied that the neurologist did not wish it. Dr. Conrad Berens asked if labyrinthine tests were made? Dr. Dodd could not definitely answer. Dr. McDannald had had a similar case in which a diagnosis of cerebellar tumor was made. Dr. Cushing did a decompression without relief; two weeks later a cyst was removed with complete recovery.

Dr. JAMES W. SMITH presented an unusual case of **Unilateral Neuroretinitis.** Negress, aged 20, came to the clinic October 4, 1923, complaining of a blind left eye for two weeks; the vision had been failing for six weeks, O.D. normal. O.S. fingers at two feet. Fundus showed papillitis 1D swelling. Midway between disk and macula there were about twenty white spots and a star-shaped figure around the macula. Tests showed the sinus and the head to be negative. On December 13th, one week later, there was marked increase in the swelling of the nerve head, with a complete star figure at the macula. On December 20th, was given K.I. and mercury inunctions. On January 17th, O.S. V. $\frac{1}{8}$. The swelling had almost subsided, and the retinal condition showed only faint striæ. Dr. Smith presents this case on account of the rapid improvement under haphazard treatment where the etiological factor could not be determined.

Dr. C. E. MCDANNALD presented a case of **Angioma of Orbit** coming on rapidly in a boy. There was a bluish tumor 3 by

2*cm* at the lower orbital margin of the left eye. Blood was aspirated, followed by a pressure bandage, but upon removal of pressure two days later the tumor returned. Dr. McDannald proposes to remove the tumor.

DISCUSSION: Dr. Somberg asked if any other tumor was present on the body? Answer: No. Dr. Buckley thinks this is an aneurism of the angular vein.

Dr. C. E. MCDANNALD presented a case of **Congenital Coloboma** of **Upper Lid repaired by plastic.**

Dr. ALFRED WIENER reported a case of **Buphthalmus** with **Neurofibromatosis** of the fifth nerve in a child two years old. Family history negative. At the age of one year the mother first noticed that the child's face was becoming distorted. At the present time the left eye presents all the appearances of buphthalmus. The upper jaw on the left side is distinctly hypertrophied and presents a number of erupting teeth. The outer portion of the upper lid is thickened. Over the frontal bone on the left side there can be felt a number of distinct cord like masses, probably hypertrophied nerve fibers. There is a pigment patch on the left shoulder and one in the groin. Wassermann negative, physical examination normal. X-ray of the skull shows the left orbit larger than right, and the left malar, denser. Dr. Wiener reviewed the literature with comments on the cases of Recklinghausen, Cabanes, Collins, Sutherland and Mayou. Due to the chronic course of the disease, Dr. Wiener pointed out that the exact pathological connection of buphthalmus and neurofibro matosis is not known. Prognosis should be guarded and the treatment confined to relieving symptoms.

Dr. MORRIS H. KAHN read a paper on **Post-Mortem Ophthalmoscopy** in which he pointed out the salient changes in the circulation of the retina occurring immediately after death. These changes consist in the formation of transverse interruptions in the red color of the vessels, giving the appearance of division or striation of the blood column. These interruptions, often only few in number are apparent chiefly in the veins and occur both near the disk and distant from it. Repeated examinations show a distinct variation in the length of one or the other striations which can be altered by pressure on the globe. After about three hours these striations are not

influenced by pressure. Dr. Kahn holds that this segmentation is a definite sign of death, and should rank clinically with the other signs of death.

MEETING OF FEBRUARY 18, 1924.

Dr. BERTHA DRAPKIN read a paper on the uses of the **Slit-lamp** and **Corneal Microscope,** calling attention to methods of use and classifications of results. After outlining the classifications of Vogt from the differentiation of pigmentation, Dr. Drapkin proceeded to enunciate the differential diagnostic points as brought out by the four methods of illumination in the classification of the pigment deposits.

Dr. CHARLES STEVENS demonstrated his camera for **Photographing** some of his many **Observations** with the **Slit-lamp microscope,** pointing out the salient features in each picture.

DISCUSSION: Dr. SCHOENBERG suggested that the Slit-lamp could be profitably used in the following: study of the circulation in the smallest conjunctival vessels; vital staining of the nerve fibers of the cornea in neurotrophic disturbances; the permeability of the ciliary epithelium as shown by the staining of the aqueous with fluorescin. The slit-lamp, in his opinion, was furthermore of great value in solving many of the daily problems in ophthalmic routine.

Dr. WHEELER told of his experiences in Vogt's clinic and the method of teaching employed there. Much was learned from the use of animals' eyes, and Dr. Wheeler suggests that all beginners with the slit-lamp make use of this method.

Dr. KEY asked if Dr. Bedell had used any vital staining of the eye, also if he could determine the predominance of pigment cells or any other elements.

Dr. Bedell in answering stated that his experience with vital staining had been too little to venture an opinion. In closing the discussion, Dr. Bedell emphasized the fact that it was not imperative to go to any place to study the slit-lamp microscopy, other than in your own office.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A meeting of the Section was held on February 8th, under the chairmanship of the Section President, Mr. A. L. Whitehead (Leeds).

Cases.

Mr. A. D. Griffith showed a case of tuberculosis arising in the optic nerve head in a man. When he first saw the patient there was no vitreous effusion, and the mass was obviously arising from the nasal half of the optic disk and the immediately surrounding retina. Since that date, the growth had passed nasally forwards into the vitreous, leaving a patch of apparently normal retina between the mass and the nerve. The man had suffered from pulmonary tuberculosis. The Wassermann was negative.

Mr. Rayner D. Batten referred to an apparently similar case which had been under his care; that also was a mass on the nasal side of the disk. This woman had a large vitreous hemorrhage at first, and the vitreous did not become clear until a year later. When it did clear, she had developed a mass on the nasal side of the disk, first in a ringed shape, like retinitis circinata. He pointed out that in the case now shown there were many vitreous opacities. In his own case the cause was not known.

Mr. Griffith also showed an infant who was brought to hospital four hours after birth. It was a forceps delivery for

placenta prævia. The right eye was luxated forwards out of the orbit, the conjunctiva was torn round the limbus, the internal rectus was ruptured, and subsequently ulceration of the cornea occurred, though the latter subsequently healed up. Now, the eye could be adducted a little, and there seemed to be a weak attachment to the globe. The optic nerve was evidently ruptured.

Miss ROSA FORD showed a woman with what was regarded as ethmoidal mucocele. The complaint was that the eyes were painful and were watering; she also had occasional momentary diplopia, and there was a chronic discharge from the left side of the nose. While she had scarlet fever, at 6 years of age, a lacrymal abscess was opened on the right side, and chronic discharge from the left nostril dated from then. At the present time a cystic swelling could be seen in the left orbit, extending above and below the internal tarsal ligament and back to the inner wall. The eye was displaced slightly upwards and forwards, and on extreme movement in any direction diplopia was experienced. There were no fundus changes, and vision in the left eye was $\frac{6}{6}$. Mr. GAY FRENCH regarded it as an ethmoidal mucocele.

Mr. J. GRAY CLEGG thought this should be attacked from the inside.

Mr. T. HARRISON BUTLER thought this might turn out to be a frontal mucocele, and that such cases should be handed over to the rhinologist to deal with. This was the more necessary as the surgeon might be faced with the need of doing a complete Killian operation. Unless quite familiar with the technique, there might result a depressed scar, or diffuse osteomyelitis.

Mr. R. AFFLECK GREEVES thought this was more likely to be a frontal than an ethmoidal mucocele; the ethmoidal cases he had seen pointed lower down.

Mr. R. LINDSAY REA pointed out that such a case might be brought to the ophthalmologist for an opinion, and he could not straightway hand it over to a rhinologist. The kind of tube used to get a skiagram of these cases was usually too soft one was required giving a 12″ spark-gap.

The PRESIDENT regarded this as a fronto-ethmoidal mucocele. He had had some experience in nasal work, and to

him this was fairly obvious. It should be attacked from the inside, from the nasal side. Clearly it was a case for the rhinologist, and there need be no scarring.

Mr. BASIL LANG read two papers on related subjects: the first entitled "A Modification of the Usual Method of Removing the Lens in the Extraction of Senile Cataract," and the second, "A Modification of the Usual Method of Needling the Lens Capsule after Cataract Extraction."

In the first of these contributions he referred to a case he was told of in which a tumor was seen in one eye, lying over the ciliary body, and this tumor was regarded as malignant. No lens could be seen behind the pupil. The eye was excised, and section showed it to be the lens; it had apparently been forced into that position when an attempted extraction was done. He remarked that the lens did not always begin to come forwards on the first pressure of the cornea below. Though he had watched many operators, he had rarely seen the lens born without some coaxing, or aided by the cystotome. The first effect of placing the curette on the lower part of the cornea was to decrease the intra-ocular volume, and he went on to explain in detail what he considered happened, from the point of view of mechanics, at each stage in the operation. He thought it would be distinctly advantageous if the hole through which the lens was to be delivered could be made as deep as possible in the antero-posterior diameter: this was his modification of the customary technique. Having completed a large section, he lacerated the capsule with the cystotome. With the patient looking down, he pressed the scoop flat on the sclera at the 12 o'clock position, with its edge in contact with the wound, and he pressed it backwards towards the center of the globe. The wound began to gape, and the vitreous forced the lens forwards against the under surface of the cornea. Further pressure increased this gaping, and the lens began to force the iris in front of it out of the wound. On further pressure the lens edge was seen to stretch the iris until it came close up to the pupillary border, and now for the first time Mr. Lang applied pressure below with the curette against the cornea. The iris was never subjected to pressure between the sclera and the lens, and apparently had not lost its tone, and the slightest touch with the repositor, or the stream from the

irrigator, caused it to return to within the eye, and the pupil assumed a central position. His modification, he submitted, rendered the operation both simple and safe. At Moorfields, before he adopted this modification, he had 5 prolapses in 43 straightforward simple extractions. He had given up using eserine drops. In the last 31 cases at the same hospital he had had only 2 prolapses, one of which should not be brought into the series, as the iris of that was atrophic. By avoiding damage to the iris, iritis did not follow the instillation of eserine. The iris did not lie flaccid and toneless in contact with the wound; rather it was taut, thus allowing any escaping aqueous to run over its surface.

In his paper on his modification for needling the lens capsule after cataract extraction, Mr. Lang said that after the lens had been removed and the eye was quiet, it was desirable to make a hole in the lens capsule, otherwise at a remote date the visual acuity would begin to be reduced. At that date the capsule would have lost its elasticity, and the operation would be difficult, as the incision would not gape, and any flap cut would fall back into its original place and continue to occlude the pupillary area. The needling was intended for its remote benefit rather than its immediate. In former days eyes were lost from one of two reasons: sepsis, or glaucoma due to the angle becoming blocked by vitreous, or by exudate from a chronic cyclitis, or from both causes. The sepsis resulted from the operation tract becoming infected with organisms from the conjunctival sac, and it could probably only occur if the needle was made to enter the anterior chamber by passing it directly through the cornea. To avoid this, the needle should be passed subconjunctivally into the anterior chamber. To make more sure, the surface of the conjunctiva could be painted at the point of entry of the needle with a 1% solution of silver nitrate. Whenever vitreous got into the anterior chamber, glaucoma was liable to occur. This was why dislocation of the lens occasionally ended so disastrously in glaucoma; for this reason he did not advise patients to have the lens removed in its capsule, as that left the vitreous unsupported. He was careful to procure a needle of suitable proportions; if the shaft of the needle was of the correct diameter, it just filled the hole and prevented escape of aqueous.

If the shaft diameter were 1, the width of the blade should be 1½. His habit was to use a Bowman's stopped needle; not a Ziegler's knife; moreover, it was rarer to find the latter with a shaft the correct size. The sooner the operation was done, the easier it was. He never divided the capsule if there was any quantity of unabsorbed lens matter. In the latter event when the eye was white, he passed the needle into the anterior chamber, rotating the shaft through 90°, moving the blade paddle-fashion to stir up any lens matter. He then had the eye hot bathed, and in a few days the lens matter had been absorbed. He described his procedure with a wealth of detail. He had the room darkened and good focal illumination, to enable one to see the gossamer-like posterior capsule. With a needle of the correct proportions none of the aqueous would escape from the anterior chamber, and the capsule could be cut three or four times if necessary.

He summed up the advantages of this modification as follows: It was easy; it avoided the possibility of sepsis; it obviated the possibility of glaucoma, and it eliminated the necessity of stirring up the vitreous.

Dr. W. H. BRAILEY (Brighton) said he agreed that in doing the extraction it was important to get rid of the sclerotic behind the lens. By not putting on pressure below, one lost the mechanical advantage of tilting the lens forward. He thought the worst cases of glaucoma were those due to the ingrowing of corneal epithelium through the wound.

Sir RICHARD R. CRUISE said it was most important, in doing cataract extraction, to produce a tilting forward of the lens. His practice was to do a simple extraction, and he invariably fully dilated the pupil. He lacerated the upper part of the capsule first with a cystotome, meeting that with a T-shaped incision coming from below upwards. The lens had then begun to present in the forward position. He then produced pressure on the posterior lip of the wound through the eyelid —he did not use an instrument for this. With a tortoise shell spoon he coaxed the lens out. The iris should be contracted into as rigid a mass as possible, so that fluid could escape round the border of it, rather than pushing a fold in front of it. In the last four years he had had two cases with a slight adhesion of the anterior surface of the iris to the wound,

and in each of those he made a small incision with a Ziegler knife in the lower part and pulled it off with a repositor.

Mr. J. GRAY CLEGG expressed his reluctance to do discission in the early stages. Most of his cases returned with excellent acuity of vision without resorting to this.

Mr. T. HARRISON BUTLER remarked that much had been said about pressure, and said that in doing cataract extraction the first thing to avoid was pressure. The operation should be so planned that the lens came out with a mere touch. The objection to the simple operation was that it demanded more pressure than did one in which iridectomy was done. He did not think there was a better lens expression than the Heidelberg. He preferred the Ziegler knife, but it could only be resharpened once if it was to retain its efficiency. Professor Ziegler was always most careful to have his knife so made that the shaft not only fitted the opening made by the blade, but was exactly parallel, so that in making cutting movements it could be slid backwards and forwards through the cornea without moving the cornea. One could examine the capsule carefully and make the incision in such wise that the gap could be made where it was wanted. He aimed to get as large an opening as he could, but why needle? When sepsis followed, he felt sure in some cases it came from the inside, that it was blood-borne. He did not know why further risks should be taken when vision was $\frac{6}{9}$ or $\frac{6}{12}$.

Mr. H. KIRKPATRICK referred to the operation now carried out in Madras.

The PRESIDENT said that after some experience he had now reverted to the combined operation, and with it he was satisfied. He agreed that if patients had $\frac{6}{6}$ or $\frac{6}{9}$ vision they might be left without needling. He did not advise needling within a few weeks of cataract extraction.

Mr. BASIL LANG, in his reply, dealt with the mechanics of the procedure and said his object was to do the least possible damage to the iris. He knew of only one case in which epithelium grew over the inner surface of the cornea. He thought the dirty eyelid ought to be kept out of the clean wound. In his last 32 extractions he had had only one prolapse, and that was due to the patient being specially sensitive to the aspirin given, and she vomited violently. He had certainly seen cases

which would have been better had they been needled. If a capsule were allowed to get into the wound, nothing would save the eye. In regard to the alleged needling failures, he would want to know how long they were made for, in what medium, how the swab was taken, and who made the cultures.

Mr. MALCOLM L. HEPBURN read a paper on **Classification of Diseases of the Choroid.** He said the nomenclature in relation to diseases of the choroid had always, in his view, been unsatisfactory; and there seemed to be no settled opinion as to the ætiology and course of the disease processes attacking the choroid. Fundus diseases ought to be classified according to the structure in which they occurred. The results of classification by position alone were that the terms retino-choroiditis, myopic choroiditis, choroidal atrophy, degenerations, holes, etc., were applied indiscriminately to all sorts of diseases, regardless of their pathological meaning, or whether they originated in the choroid or the retina. In this paper he dealt with choroid only. All such conditions were associated with pigmentary disturbances, but in solely retinal affections no such changes occurred. The American Encyclopædia of Ophthalmology contained 26 forms of choroidal inflammation which seemed to require separate headings.

The classification he now put forward fell into five groups: 1. Inflammatory; 2. Vascular; 3. Degenerative; 4. Congenital; 5. New Growths. The need for discriminating between the first two groups was, that in the recent stage the treatment was quite different. At present the term retino-choroiditis was used to describe both acute and scarred forms, whereas it really implied an acute inflammation. The clinical features associated with inflammatory diseases of the choroid were vitreous opacities, and sometimes keratitic deposits in the acute stage, with scotomata in the usual field, of different shapes and extent, and which remained permanent in the scarred stage. The acuity of vision was suddenly reduced to considerably below normal, but might recover completely when the inflammatory exudate had become absorbed, unless the macula happened to be the part involved. He contrasted this with a typical case of choroidal affection due to vascular changes—primary pigmentary degeneration of the retina. In the latter there was the typical bone-corpuscle-shaped

arrangement of pigment in the mid-peripheral region, scattered over an otherwise normal looking fundus, and the cause had been acknowledged to be the altered condition of the choroi-capillaris. Cases were met with in which the bone-corpuscle-shaped pigment was scattered over a fundus which was not normal, and where evident changes in the choroid had taken place previously, resulting in the larger vessels becoming affected. In this way the choroidal blood supply was cut off, and the same effect was produced on the retina as in primary pigmentary degeneration. These were cases of pigmentary degeneration secondary to disease of the choroid. A hæmorrhage from the choroidal blood vessels would sometimes be so extensive as to wash out the pigment mechanically from the pigment epithelial layer, when it was seen indistinctly mixed up with the blood. When the latter became absorbed, these small masses of pigment were left high and dry, giving a granular appearance to the disturbed area, but there was no proliferation.

In degenerations, such as the typical Tay's choroiditis, the pigment was merely pushed aside by the hyaline masses; no proliferation of pigment could be seen, only small white or yellowish white patches, with an even pigmented border. Tay's choroiditis was now known to be a hyaline degeneration of the membrane of Bruch.

From the considerations set out in the paper, the author thought the following conclusions could be drawn:

1. Where there was an œdematous patch accompanied by a sudden lowering of the visual acuity, vitreous opacities and keratitic precipitates, followed by the formation of fibrous tissue of varying amount, with many coarse masses of pigmentation, the causal condition was of inflammatory origin.

2. Where the pigmentary changes were of a fine granular type, and the choroidal disturbance was of a somewhat indefinite character, accompanied by a varied and gradual reduction of vision with no vitreous opacities and no keratitic precipitates, it was of vascular origin.

3. Where white or whitish-yellow areas were found, surrounded by an even, well-defined border of pigment, not excessive in quantity, with no choroidal disturbance round it, it was a hyaline degeneration, probably of the membrane of Bruch.

4. When there was a pearly-white patch, with no vessels crossing the floor of it, and a narrow fringe of pigment round a well-defined margin, and with no choroidal disturbance beyond the actual defect, probably it was of congenital origin.

He said the characteristic features of a new growth were the raised choroidal swellings, often with a well-defined, non-edematous border, and any pigment present was indefinitely mixed up with the main mass. Frequently there was an ordinary detachment, a separation between the neuro-epithelial layer and the pigment epithelial layer, somewhere in the neighbourhood of the growth. The diagnosis was helped by the presence of new vessels.

He showed, by the epidiascope, drawings illustrative of the points he was seeking to establish. Dr. Batten, he said, had attempted to associate many diseases of the macula with affections of the brain, causing various mental disorders, and that gentleman associated the two together as a disease of the nervous system, analagous to amaurotic family idiocy. That Mr. Hepburn looked upon as a stretching of the analogy too far.

Mr. RAYNER BATTEN claimed a special place in classifications for diseases of the macula, as that region was singled out from the rest of the fundus anatomically, physiologically and clinically. True, it contained both choroid and retina, and therefore was liable to be involved in diseases affecting either of those structures. The macula itself was avascular, and was surrounded by a network of terminal vessels. It was liable to special involvement in some diseases, and practical exemption in others. In the latter were syphilis and tubercle. In glaucoma this area might survive to the end, yet in myopia it seemed to be singled out for destruction. Macular disease was so markedly symmetrical that if symmetry was absent in a case, he felt doubts as to whether it should be classed as a macular case.

The time for the meeting having expired, the author did not reply.

H. DICKINSON.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London, and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M.D., F.A.C.S., New Rochelle, N. Y.

(*Continued from January issue.*)

VIII.—THE SENSE OF SIGHT, REFRACTION AND ACCOMMODATION.

111. Clausen. **Heredity of red green blindness.** *Ver. d. Ærtze in Halle*, June 19, 1921.

112. Comberg. **The so-called snowy landscape phenomenon.** *Zeitschrift f. Sin-nesphysiol.*, liii., p. 179.

113. Deyo, Barbara V. **Monocular and binocular judgment of distance.** *American Journal of Ophthalmology*, May, 1922.

114. Dimmer. **Light sensation with closed lids.** *Archiv f. Ophthalm.* cv., p. 794.

115. Duane, A. **Studies in monocular and binocular accommodation with their clinical applications.** *American Jour. of Ophthalm.*, November, 1922.

116. Engelking. **The pupillary reaction in congenital total color blindness.** *Klin. Monatsbl. f. Augenheilkunde*, lxvi., p. 707.

117. Ferree, C. E. and Rand, G. **Perimetry; variable factors influencing the breadth of the color fields.** *American Journal of Ophthalmology*, November, 1922.

118. Froehlich, F. W. **The influence of adaptation to light and dark on the course of periodic after images.** *Zeitschr. f. Physiol. d. Sinnesorgane*, liii., p. 79.

119. Hagen. **Ætiology of the transitory hypermetropia in diabetes mellitus.** *Archiv f. Ophthalmologie*, cv., p. 243.

120. Hanssen. **Genesis of myopia.** *Klin. Monatsbl. f. Augenheilk.*, lxvii., p. 171.

121. Hirsch, G. **Cause of myopia.** *Wochenschr. f. Therapie u Hygiene d. Auges*, xxiv., No. 9.

122. Kleczkowsky. **Physiology and pathology of adaptation of the eye to dark.** *Archiv f. Augenheilkunde*, lxxxv., p. 289, and lxxxviii., p. 253.

123. LANDOLT, E. **The angle alpha.** *American Jour. of Ophthalmology*, May, 1922.

124. LARSEN. **Microscopic preparations of a monochromatic eye.** *Ophthalm. Gesellsch*, Vienna, August 4 to 6, 1921.

125. LEVINSOHN. **Accommodation in aphakia.** *Berl. klin. Wochenschrift*, xxxii., p. 915.

126. LÖWENSTEIN. **The influence of limitation of light upon the vision.** *Archiv f. Ophthalmologie*, cv., p. 844.

127. LUEDDE, W. H. **Amblyopia ex anopsia and recovery of vision.** *American Journal of Ophthalmology*, June, 1922.

128. MARQUEZ. **Frequency of biastigmatism.** *Ophthalmic Society of Vienna*, August 4–6, 1921.

129. SCHANZ. **Injury of the retina by ultraviolet light.** *Archiv f. Ophthalmologie*, cvi., p. 171.

130. SCHANZ. **Vision.** *Ophthalmic Society of Vienna*, August 4–6, 1921.

131. SCHANZ. **Color vision.** *Muench. med. Wochenschr.*, xliii., p. 1390.

132. SITTIG. **Disturbances of the color sense in aphasics.** *Monatschr. f. Psychiatr. u. Neurol.*, xlix., pp. 63 and 159.

133. TEFFT, L. E. AND STARK, ELIZABETH K. **Speed accommodation as a practicable test for fliers.** *American Journal of Ophthalmology*, May, 1922.

134. WERNICKE, O. **Sclerosis disseminate as the cause of progressive myopia and of diabetes.** *Buenos Ayres*, 1921.

135. ZEEMANN. **Adaptation in a case of syphilitic neuritis retinæ.** *Archiv f. Ophthalmologie*, cvi., p. 1.

SCHANZ (130, **Vision**) advances a new theory of vision in which the sensibilators play a great part in the action of light upon the albumin. Negative electrons are thrown out which seize upon the molecules of albumin and produce changes. The rods and cones cannot absorb visible light rays, it is the pigment epithelium which throws out the negative electrons, and these agitate the rods and cones. Albinos see because traces of pigment are present.

DEYO'S (113, **Monocular and binocular judgment of distance**) studies show great differences in depth perception made with binocular and monocular tests, and seem to prove that this faculty is dependent upon the binocular parallax.

LUEDDE (127, **Amblyopia ex anopsia**) sides with those who think amblyopia in the squinting eye is acquired and offers the usual arguments. He relates a number of cases in which he apparently improved the vision by excluding the better eye over long periods and thinks that this treatment should

not be omitted. However there are those who cannot recall cases in which the vision deteriorated after the onset of the strabismus and cases are not infrequently seen when hypermetropia and amblyopia exist with fusion and without strabismus.

SCHANZ (129, **Injury of the retina by ultraviolet light**) has found among glassblowers, bakers, and workmen in other callings who have much to do with bright electric lights, in addition to subjective troubles a reduction of the adaptation to dark. The remarkably large number of hemeralopes discovered in the war may be the best proof that insufficient attention is paid during peace to the harm done to adaptation by ultraviolet light.

LOEWENSTEIN (126, **Influence of limitation of light upon the vision**) says that when a myope holds an object in front of one eye inside of its near point and fixes on a test type outside its far point a portion of the test type which is seen in the shadow edge of the object appears darker and more sharply defined, and also somewhat displaced. This he ascribes mainly to a diminution of the dispersion circles by the outline of the object, but this alone does not explain the improvement of the retinal image. The reduction of the quantity of light in the shadow plays a part, and so does the function of contrast. Contrast contracts the area of aberration and brings into prominence only a certain middle zone, not the marginal zone. With a broad shadow edge the best retinal image appears in the dense part of the marginal shadow.

DIMMER (114, **Light sensation with closed lids**) says that the intensity of the light source plays an important part in the localization. When it is strong it is projected correctly to the nasal side, while with a weak light it is located only on the temporal side. Examination of several enucleated eyes makes it probable that a false projection may thus be obtained.

COMBERG (112, **The so-called snowy landscape phenomenon**) presents the following explanation why a field of snow often appears considerably brighter than the heavens from which it receives the light. Three factors take part. (1) The cloudy sky appears darker at the horizon, where it touches the field of snow and is commonly compared with it, than at the zenith; the eye receives only a fraction of the light from the

sky at the horizon, while it receives nearly all of that from the surface of the snow; (2) the cutting off by the upper lid of the surface of the sky above the horizon; (3) occasionally the field below the horizon may appear the brighter through the effect of contrast and adaptation. By comparison of the reflections from two mirrors he was able to demonstrate that the reflection from the sky was the brighter.

FROEHLICH (118, **Influence of adaptation to light and dark on the course of periodic after images**) finds that the reaction of our visual apparatus to brief lighting is essentially delayed by adaptation to dark. The varying involvement of different segments of the retina by the adaptation gives rise to the characteristic changes of form of after images. The excitability of the fovea centralis is less in all conditions of adaptation than that of the periphery, and the retina has a paracentral zone in which the amplitude of adaptation corresponds to that in the periphery, but with a much less rapidity. In the course of an adaptation to dark after a previous adaptation to light there are three phases, first a slight rise, second a sharp rise and third a gradual rise of sensitiveness. The elements of the retina sensitive to color show an amplitude of adaptation increasing from the fovea toward the periphery.

KLECZKOWSKY (122, **Physiology and pathology of adaptation of the eye to dark**) had the patient fix on a red light and then used for testing a small electric light, the strength of which was regulated by means of a rheostat, which gave the candle power employed. He found that the adaptation in the region of the yellow spot is less than at the periphery, and that this area appears larger as the distance of the observer increases. In congenital amblyopia he demonstrated a circular circumfoveal scotoma for weak light, the size of which increases with diminution of the intensity of the light. The light sensitiveness of excentric portions of the retina at the same time is undisturbed. In diseased eyes he found that in every case in which a scotoma could be demonstrated by Bjerrum's method its existence could be proved by testing in the dark. He also found that the size of the scotoma in which a disturbance of adaptation exists depends upon three things, the size of the pathological focus, whether the disease is old or fresh, and the light strength of the test object.

ZEEMANN (135, **Adaptation in a case of syphilitic neuritis retinæ**) at first found the sensitiveness very slight at each point of time of the examination, while the amplitude of adaptation apparently followed a normal course. During convalescence the sensitiveness increased, abruptly at first and then more slowly until it reached normal. A study of the field explains this course. There was present at first a central scotoma and the rapid improvement of this caused the abrupt rise in sensitiveness.

CLAUSEN (111, **Heredity of red green blindness**) describes four families of five generations each, in which red green blindness was hereditary.

ENGELKING (116, **The pupillary reaction in congenital color blindness**) made the following observations in a single case. The reaction of the pupil to light was completely absent in extreme adaptation to light, and gradually approached the normal with increasing adaptation to dark. After repeated irradiation the pupils did not seem to dilate as widely as they should. Possibly also the contraction was somewhat slower than normal.

FERREE and RAND (117, **Variable factors influencing the breadth of the color fields**) present the first of a number of papers on perimetry which promise to standardize our knowledge of this subject. This paper deals with the effect of the varying intensity of illumination of the test object on the extent and the shape of the fields. The range of sensitivity of the retina from the center to the periphery is very great being 900 times for red, 3380 times for green. There is also great irregularity in the decrease of sensitivity from the center to the periphery for different meridians tested not only for different colors but also in the ratio for different colors so that the fields may cross. Under sufficiently strong illumination all colors except green have a field as wide as that for white. The impression gained from reading this paper is that the methods generally employed for taking color fields are decidedly unreliable.

SCHANZ (131, **Color vision**) holds that electrons are thrown out from the pigment epithelium in consequence of the absorption of light and seized upon by the rods and cones as antennæ. Electrons of various rapidities correspond to the different

wave lengths of light. Thus arises the perception of pure colors. With regard to impure colors he thinks that the electrons which find themselves simultaneously on the track to the central organ mutually influence their rapidity and gradually assume an average rapidity. His explanation of Purkinje's phenomenon is that the electrons which red light throws out from the pigment epithelium can no longer stimulate in reduced illumination because of their less swiftness.

SITTIG (132, **Disturbance of the color sense in aphasics**) reports three cases, one of gun shot of the brain, one of thrombotic softening, and one of tumor, all of which had aphasia and disturbance of the color sense.

It is necessary in these cases to differentiate those in which the color sense itself remains intact, but the patients cannot name colors rightly and do not understand the names; these have an amnesia of the names of colors. Others are unable to distinguish the colors themselves. Both conditions are associated with such other complications as alexia, mental blindness, or amnesic aphasia.

LARSEN (124, **Microscopic preparations of a monochromatic eye**) found no fewer cones than normal. In the fovea the cones were short and thick with short outer limbs, laterally mixed with short rods. Hence the theory that rods alone are present in total color blindness is erroneous.

HAGEN (119, **Ætiology of transitory hypermetropia in diabetes**) concludes that hypermetropia does not appear until after treatment for the diabetes has been instituted and that it is probably not a true complication of the disease, but is a consequence of the therapy.

LANDOLT (123, **The angle alpha**) explains that this angle is formed by the visual line which passes through the object of fixation, through the two nodal points to the macula and the major axis of the corneal ellipsoid which passes through the anterior nodal point. The angle has been defined as that made by the visual line and the optic axis, but this is incorrect.

MARQUEZ (128, **Biastigmatism**) measures the corneal astigmatism by means of the ophthalmometer, places the corresponding cylinder in front of the eye, measures the remaining astigmatism by means of the astigmatic chart and corrects it

with a second cylinder. The combination of cylinders is then reduced to a sphero-cylindrical formula.

HANSSEN (120, **Genesis of myopia**) found that the enlarged globe in a case of high myopia extended far back into the orbit pressing the optic nerve into a well marked S loop. Such a condition as this proves all theories incorrect which ascribe nearsightedness to a pulling upon the posterior segment of the eyeball by the optic nerve.

HIRSCH (121, **Cause of myopia**) gives as the necessary precondition an abnormal extensibility of the coats of the eye. Anatomical studies have shown that the posterior portions of the sclera and choroid are atrophic. This region is supplied by the short posterior ciliary vessels. The fact that corneal scars and myopia often coincide suggests that the disease underlying the atrophy of the sclera is tuberculous, like the scrofulous keratitis which underlay the corneal scars. He found that all the persons examined by him who had progressive myopia were strongly positive to Koch's tuberculin test, and that the vision improved after the first injection. His idea is that the toxin of tuberculosis works harm to the sclera and choroid, leading to posterior staphyloma and central choroiditis, and he ascribes the detachment of the retina to the same cause. He thinks the transmission of tuberculosis in schoolrooms to be very common.

WERNICKE (134, **Sclerosis disseminata the cause of progressive myopia**) ascribes both myopia and glaucoma to multiple sclerosis. This view is based on the changes in the papilla, retina and choroid. If the sclerotic foci are situated in the ciliary region, and especially in the iris, premonitory symptoms of glaucoma appear, if farther back detachment is threatened. Through similar conclusions from analogy he is led to ascribe diabetes to multiple sclerosis.

DUANE (115, **Studies in monocular and binocular accommodation with their clinical applications**) has added the results of the examination of 2000 eyes to those of the 2200 already reported. The observations on the newer cases substantially confirm those made ten years ago. Tables and curves are presented showing the maximum and minimum limits of monocular accommodation as well as the mean value for all ages. Values are also plotted for binocular accommodation.

The following figures are taken from the table recording normal accommodation for each year:

Age	*Minimum*	*Mean*	*Maximum*
10	11.1	13.4	15.7
20	8.9	11.1	13.4
30	6.5	8.7	10.8
40	3.4	5.8	7.9
45	1.9	3.6	5.9
50	1.	1.9	3.2
55	0.8	1.3	1.9
60	0.7	1.2	1.7
64 to 70	0.6	1.	1.6

Binocular accommodation is uniformly greater than monocular, from 0.2 D to 0.4 D being added after the age of 32. He thinks the contraction of the pupil is a factor of little importance in accommodation. The activity of the ciliary muscle decreases together with the elasticity of the lens as the age advances so that little is left in the aged. Subnormal accommodation may be either lenticular or ciliary, the former being stable and causing no reflex symptoms, the latter, however, often causing marked asthenopia. The accommodation fails equally in the two sexes.

TEFFT and STARK (133, **Speed accommodation as a practicable test for fliers**) have studied the reactions of applicants by means of the apparatus of Ferree and Rand and present a table showing the mean time in various groups formed on the basis of a complete eye examination with the view to determine the qualifications for flying. Rapid accommodation is considered essential to safety.

LEVINSOHN (125, **Accommodation in aphakia**) reports the case of a man 47 years old operated on for cataract in each eye. Perfect vision was obtained in each eye with the correcting glass, and the patient could read the finest print with the same glass. Levinsohn thinks that a change took place in the form of the eye under the influence of muscle pressure, probably a stretching of the posterior part of the eyeball.

(To be continued.)

BOOK REVIEWS.

II.—**The Relative Position of Rest of the Eyes and the Prolonged Occlusion Test.** By F. W. MARLOW, M.D., M.R.C.S. (Eng.), F.A.C.S. F. A. Davis Company, Publishers.

In this little book Dr. Marlow has assembled extensive clinical data supporting his well-known theses on the diagnosis and treatment of motor anomalies. A large number of cases which he has followed over a series of years are adduced to prove his belief that by occlusion of one eye kept up continuously for a week or longer the eyes assume the position of rest, and thus anomalies, otherwise obstinately latent, are revealed. The method is surely drastic; yet if it attains with certainty the result desired—*i.e.* an absolute diagnosis—it is for the patient worth all the time and trouble spent upon it.

As corollaries to this proposition, the author contends against the usefulness of muscle exercises and for the usefulness of prisms for constant wear.

Dr. Marlow's arguments are presented in a scientific spirit and with eminent fairness and moderation. They certainly deserve serious consideration, and his diagnostic method should receive thorough trial in appropriate cases. Whether it is necessary to apply it in any but a rather small minority of the cases of motor anomalies is a point on which the reviewer confesses himself still unconvinced. The question in any case is one to be solved by experience, not by theoretical reasoning. This also applies to the corollaries above mentioned. Here, on the whole, the reviewer's experience has been the reverse of Dr. Marlow's, for he has in repeated instances found muscle exercises to be beneficial, and has seen not a few cases in which the continued use of prisms, at first apparently helpful, has later ceased to work benefit or has even seemed to work harm.

A. D.

III.—**Applied Pathology in Diseases of the Nose, Throat and Ear.** By JOSEPH C. BECK, M.D., F.A.C.S. Published by the C. V. Mosby Company, 508 North Grand Boulevard, St. Louis, U. S. A. Price $7.50.

This is a handy volume of 280 pages, profusely illustrated with excellent cuts (there are 268 original illustrations). In spite of the author's statement that he has limited the book to cases coming under his personal experience, it is quite comprehensive.

The author states that his reason for writing a book on the Applied Pathology of the Nose, Throat, and Ear, is his belief that the student may derive greater benefit by applying the pathological entities to etiology, symptoms, diagnosis and prognosis, and that thereby he may arrive at a rational basis for treatment, exclusive of strictly surgical interventions. In his opinion the fundamental object in the analysis and management of a case should be a definite knowledge of the underlying changes present, and this thought has been kept uppermost throughout the book.

The close relation of diseases of the eye to those of the nasal accessory sinuses and the tonsils should render this book of especial interest to ophthalmologists. It should also appeal to beginners in oto-laryngology and to physicians and surgeons who occasionally invade this field.

A. H. T.

IV.—**International Clinics.** No. III. Thirty-third Series. J. B. Lippincott Co., Philadelphia.

In this volume may be found a good collection of articles on Diagnosis and Treatment, Morbid Psychology, Pædiatrics, Medicine, Surgery, and Legal Medicine by well-known authors. Only one of these makes any mention of the eye, and that is an article by Dr. W. Moore Thompson, entitled "Practical Hints in the Care and Treatment of the Eye, Ear, Nose, and Throat."

With reference to Dr. Thompson's advice in the treatment of eye diseases, we think that much better advice may be obtained from any good text-book on Ophthalmology; for example, in examining a child's eye, the doctor advises that the child be held on the nurse's lap with its head toward the nurse's body, its feet toward the examiner. Ophthalmologists usually

employ the opposite method. In the treatment of ophthalmia a solution of permanganate of potassium "of a delicate pink color" is advised, which certainly permits of a wide variation in the strength of the solution. Following this the doctor employs a 20% solution of silver nitrate, which is much stronger than most ophthalmologists would dare to use.

A. H. T.

V.—**International Clinics.** No. I. Thirty-fourth Series. J. B. Lippincott Co., Philadelphia.

This volume contains the usual variety of excellent articles chiefly of interest to the general practitioner. A special feature is a Symposium on the New Born by five well-known specialists in this line of work.

The following contributions should be of interest to ophthalmologists—"Exophthalmic Goiter," by Dr. Lewellys F. Barker, "Some Aspects of Migraine," by Dr. A. H. Gordon, and "Medical Aspects of Workmen's Compensation Laws," by Dr. Frank L. Rector.

A. H. T.

CORRESPONDENCE.

EDGBASTON,
BIRMINGHAM, ENGLAND.

DEAR DR. KNAPP:

In the excellent résumé of my paper on the "Practical Value of the Slit-lamp" which appears in the May number of the ARCHIVES on page 288, there is a rather serious mistake which I would ask you to have corrected. I advised members of the society to avoid at all costs the combined Slit-lamp and simplified Gullstrand. It is a most impossible form of slit-lamp, valueless for both purposes, for apart from the fact that it gives a bad beam of light, no one has time to keep changing the movement and readjusting it.

Yours sincerely,
T. HARRISON BUTLER.

May 15, 1924.

BALTIMORE, May 16, 1924.

TO THE EDITOR.
DEAR SIR:

In a recent issue of the ARCHIVES, there appears an article by Mr. T. Harrison Butler "On the Visibility of the Actual Blood Stream with the Ordinary Loupe."

Records of such observances have indeed not appeared in the literature for a very long time until interest in the subject had been reawakened by the corneal microscope and slit-lamp.

However Mr. Butler is not quite the first one to be able to see the blood stream with the ordinary loupe, as in my first ophthalmological publication which appeared in Hirschberg's *Centralblatt* in February, 1888, I recorded identical observations made with the Hartnack loupe.

It is indeed pleasing to meet such old friends.

Very truly yours,
HARRY FRIEDENWALD.